JUN 30 1913

VOL. CXLVI, No. 1 JULY, 1913 No. 496

THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES

EDITED BY GEORGE MORRIS PIERSOL, M.D.

THE OCCURRENCE OF ANKLE-CLONUS WITHOUT GROSS DISEASE OF THE CENTRAL NERVOUS SYSTEM. By WILDER TILESTON, M.D. 1

THE RATIONAL TREATMENT OF SURGICAL OR NON-PULMONARY TUBERCULOSIS. By JOHN B. HAWES, 2d, M.D. 10

MYOCARDIAL HYDROTHORAX, WITH REPORTS OF CASES. By JAMES M. AND... M.D., LL.D. 15

CONGENITAL ATRESIA OF THE DUODENUM. By ARCHIBALD L. McDONALD, A... M.D. 28

RECOVERY FROM TUBERCULAR MENINGITIS, WITH REPORT OF CASE. By ROBERT L. PITFIELD, M.D. 37

AN INTENSIVE STUDY OF THE EPIDEMIOLOGY OF PELLAGRA. REPORT OF PROGRESS. By JOSEPH F. SILER, B.S., M.D., and PHILIP E. GARRISON, A.B., M.D. . . 42

THE DIAGNOSIS OF INFLAMMATION IN THE ANTERIOR OCULAR SEGMENT. By B. F. BAER, JR., B.S., M.D. 67

EPIDIDYMITIS DUE TO THE COLON BACILLUS. By WALTER S. REYNOLDS, M.D. 72

THE RATIONAL TREATMENT OF TETANUS, WITH A REPORT OF TWENTY-THREE CASES FROM THE EPISCOPAL HOSPITAL, PHILADELPHIA. By ASTLEY PASTON COOPER ASHHURST, M.D., and RUTHERFORD LEWIS JOHN, M.D. 77

ISOLATED SCLEROTIC INVOLVEMENT OF THE MITRAL VALVE. By ROBERT N. WILLSON, M.D. 118

LIBRARY OF CONGRESS JUL 1 1913 PERIODICAL DIVISION

OWNERS AND PUBLISHERS

Philadelphia LEA & FEBIGER New York

COPYRIGHT 1913, BY LEA & FEBIGER. ALL RIGHTS RESERVED. ENTERED AT THE POST-OFFICE AT PHILADELPHIA AS SECOND-CLASS MATTER

PUBLISHED MONTHLY. SUBSCRIPTION, FIVE DOLLARS A YEAR, POSTPAID

LEA & FEBIGER

NEW AND FORTHCOMING BOOKS

TOROK & GROUT'S SURGERY OF THE EYE. JUST READY
See page 3.

LOCKWOOD ON DISEASES OF THE STOMACH. JUST READY
See page 2.

PRICE'S HYGIENE FOR NURSES.
JUST READY See page 4.

STARR ON NERVOUS DISEASES.
NEW (FOURTH) EDITION. JUST READY See page 4.

POTTS on NERVOUS AND MENTAL DISEASES. NEW (THIRD) EDITION. JUST READY
See page 2.

SMALLWOOD'S BIOLOGY. JUST READY
See page 3.

TAYLOR ON DIGESTION AND METABOLISM. See page 6

STARLING'S PHYSIOLOGY. See page 14

JACKSON AND McMURTRY ON DISEASES OF THE HAIR. See page 14

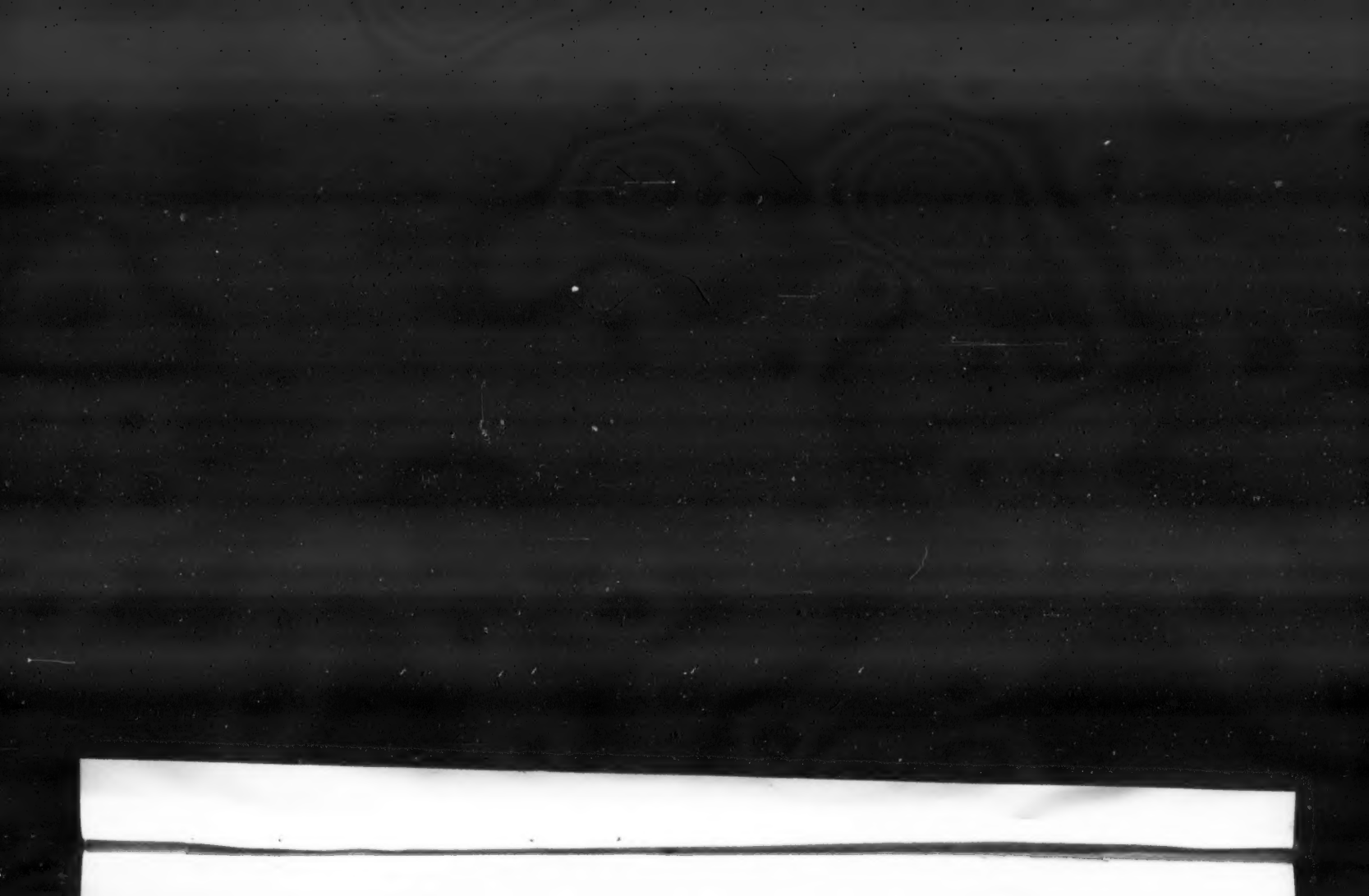
SIMON AND BASE'S MANUAL OF CHEMISTRY. TENTH EDITION
See page 7

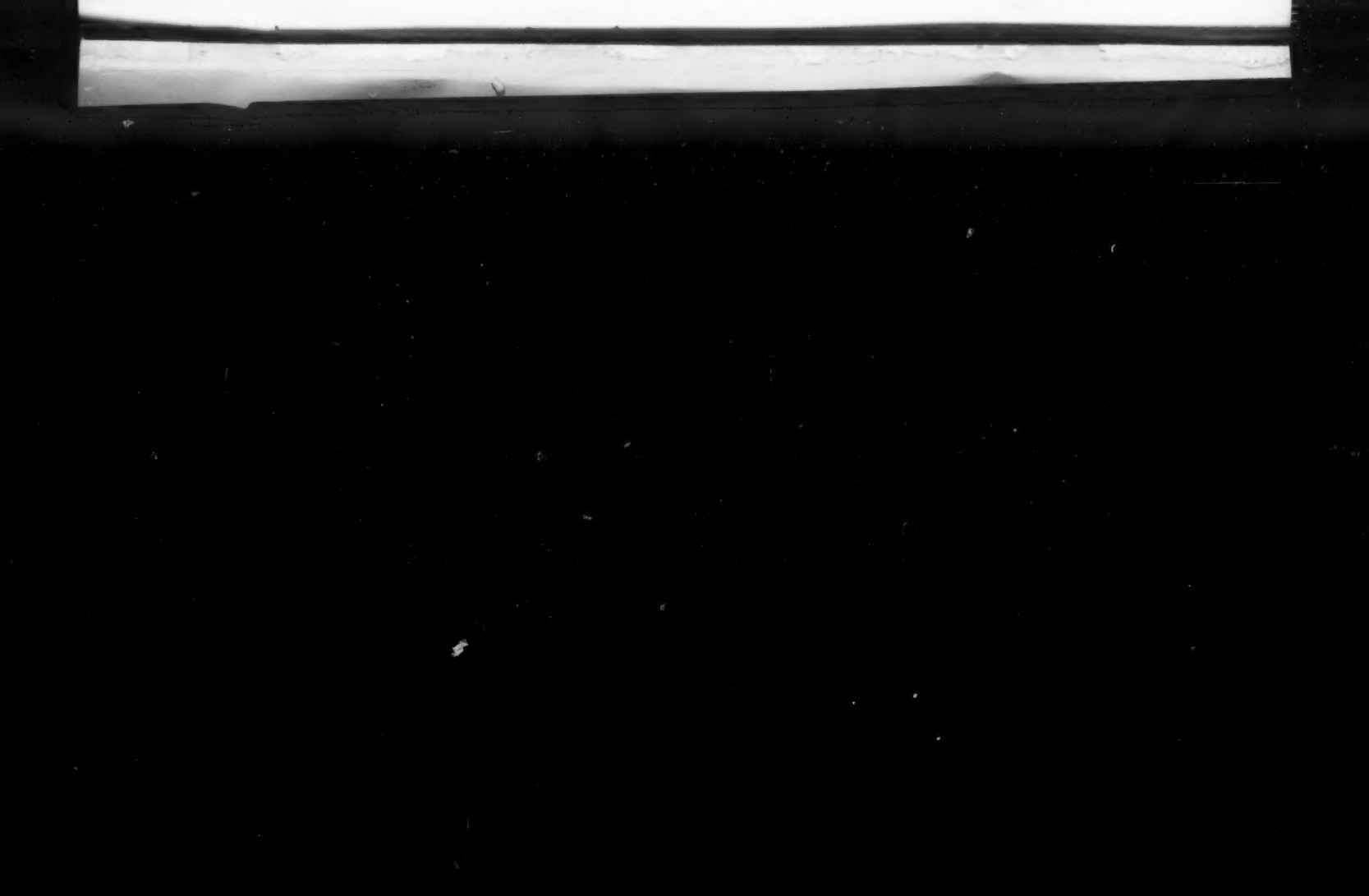
VAUGHAN ON PROTEIN POISONS

MAIN OFFICE
706-8-10 Sansom St.
PHILADELPHIA

LEA & FEBIGER
PUBLISHERS

N. Y. OFFICE
2 W. 45th Street
NEW YORK





CONTENTS

ORIGINAL ARTICLES

The Occurrence of Ankle-clonus without Gross Disease of the Central Nervous System 1

By WILDER TILESTON, M.D., Assistant Professor of Medicine, Yale University Medical School, New Haven, Connecticut.

The Rational Treatment of Surgical or Non-pulmonary Tuberculosis . 10

By JOHN B. HAWES, 2D, M.D., Director, Tuberculin Department, Assistant Visiting Physician, Massachusetts General Hospital; Secretary, Board of Trustees Massachusetts Hospitals for Consumptives.

Myocardial Hydrothorax, with Reports of Cases 15

By JAMES M. ANDERS, M.D., LL.D., Professor of Medicine in the Medico-Chirurgical College of Philadelphia.

Congenital Atresia of the Duodenum 28

By ARCHIBALD L. MCDONALD, A.B., M.D., Attending Physician, to Children's Home, Duluth, Minnesota.

Recovery from Tubercular Meningitis, with Report of Cases 37

By ROBERT L. PITFIELD, M.D., Physician to St. Timothy's and Germantown Hospitals, Philadelphia.

An Intensive Study of the Epidemiology of Pellagra. Report of Progress 42

By JOSEPH F. SILER, B.S., M.D., Captain, Medical Corps, United States Army, and PHILIP E. GARRISON, A.B., M.D., Passed Assistant Surgeon, United States Navy.

The Diagnosis of Inflammation in the Anterior Ocular Segment . . . 67

By B. F. BAER, JR., B.S., M.D., Assistant Ophthalmic Surgeon, Eye Dispensary of the Hospital of the University of Pennsylvania, Philadelphia.

Epididymitis Due to the Colon Bacillus 72

By WALTER S. REYNOLDS, M.D., Chief of the Clinic of Genito-urinary Surgery in the Medical Department of Columbia University, New York.

The Rational Treatment of Tetanus, with a Report of Twenty-three Cases from the Episcopal Hospital, Philadelphia 77

By ASTLEY PASTON COOPER ASHHURST, M.D., Instructor in Surgery in the University of Pennsylvania; Associate Surgeon to the Episcopal Hospital, etc., and RUTHERFORD LEWIS JOHN, M.D., Resident Physician, Episcopal Hospital.

Isolated Sclerotic Involvement of the Mitral Valve 118

By ROBERT N. WILLSON, M.D., of Philadelphia.

REVIEWS

Progressive Medicine. A Quarterly Digest of Advances, Discoveries, and Improvements in the Medical and Surgical Sciences. Edited by Hobart Amory Hare, assisted by Leighton F. Appleman, M.D. . . 124
Appendicitis. By John B. Deaver, M.D., Sc.D., LL.D. 125
Augustus Charles Bernays. A Memoir. By Thekla Bernays 128
The Pathology of the Living, and Other Essays. By B. G. A. Moynihan, M.S. (London), F.R.C.S. 129
Surgical Operations with Local Anesthesia. By Arthur E. Hertzler, M.D. 129
On the Physiology of the Semicircular Canals and Their Relation to Sea Sickness. By Joseph Byrne, A.M., M.D., LL.B. 130
The Surgical Treatment of Locomotor Ataxia. By L. N. Denslow, M.D. 131
Handbook of Mental Examination Methods. By Shepherd Ivory Franz, Ph.D. 132
The Therapy of Syphilis. By Paul Mulzer, M.D. 132

PROGRESS OF MEDICAL SCIENCE

MEDICINE

UNDER THE CHARGE OF

W. S. THAYER, M.D., AND ROGER S. MORRIS, M.D.

Alimentary Galactosuria in Experimental Phosphorus Poisoning . . . 133
The Bradycardia of Lead Colic 133
Acetone and Diacetic Acid 134
Syphilitic Aortitis 134
Congenital Family Steatorrhea 135
On Uric Acid Excretion in Hypophyseal Disease 135
Autoserotherapy and Absorption of Ascites 135
On the Presence of Typhoid Bacilli in the Mouth of Typhoid Fever Patients and Typhoid Convalescents 136
Blood Findings in Adiposity 136

SURGERY

UNDER THE CHARGE OF

J. WILLIAM WHITE, M.D., AND T. TURNER THOMAS, M.D.

A Procedure for Wide Extirpation of Cancer of the Prostate 137
Traumatic Epilepsy after Head Injuries in the Japanese-Russian War . 138
Treatment of Spontaneous Gangrene of the Extremities 139
Paralysis of the Phrenic Nerve from the Employment of Kulenkampff's Brachial Plexus Anesthesia 139
The Extended Abdominal Radical Operation for Cancer of the Uterus . 140

THERAPEUTICS

UNDER THE CHARGE OF

SAMUEL W. LAMBERT, M.D.

Syphilitic Disease of the Aorta . . . 141
Action of Benzol on Leukemia . . . 141
Treatment of Leukemia with Benzol . . . 142
Experiences with Neosalvarsan . . . 142
The End-results of the Abortive Treatment of Syphilis with Salvarsan . . . 142
Cholesterin in Paroxysmal Hemoglobinuria . . . 142
The Treatment of Amebic Dysentery with Subcutaneous Injections of Emetin Hydrochloride . . . 143
Amebic Abscess of Liver Treated Successfully by Emetin . . . 143

PEDIATRICS

UNDER THE CHARGE OF

LOUIS STARR, M.D., AND THOMPSON S. WESTCOTT, M.D

Chronic Infective Endocarditis . . . 144
Diphtheritic Paralysis . . . 145
Summer Heat and Summer Diarrhea . . . 145

OBSTETRICS

UNDER THE CHARGE OF

EDWARD P. DAVIS, A.M., M.D.

Albuminuria Likely to Recur in Successive Pregnancies . . . 146
Abderhalden's Serum Test for Pregnancy . . . 147
Puerperal Eclampsia . . . 147
Unusual Fertility in Syphilitic Patients, with Anomalous Involvement of the Child . . . 147
The Condition of the Blood Serum of Mother and Fetus in the Pyelitis of Pregnancy . . . 148
Subcutaneous Symphysiotomy . . . 148

GYNECOLOGY

UNDER THE CHARGE OF

JOHN G. CLARK, M.D.

Intestinal Obstruction Due to Retroverted Uterus . . . 149
Prognostic Value of Leukocyte Count in Pelvic Suppurations . . . 150
Callous Ulcer of the Bladder . . . 150

DISEASES OF THE LARYNX AND CONTIGUOUS STRUCTURES

UNDER THE CHARGE OF

J. SOLIS-COHEN, M.D.

Three Fatal Cases of Pneumococcal Infection of the Throat 151
Abnormal Epistaphylian Tonsil 152
A Case of Mixed Tumor of the Soft Palate 152
Perforation of the Nasal Septum Due to Topical Action of Cocaine . . 152
Chondrosarcoma of Nasal Passages 152
Bronchial Asthma Cured by Operations in Rhinopharyngeal Respiratory Tract 152
Topical Anesthesia in Sinus Operations 153

HYGIENE AND PUBLIC HEALTH

UNDER THE CHARGE OF

MILTON J. ROSENAU, M.D., AND MARK W. RICHARDSON, M.D.

Chlorinated Lime in Sanitation 153
The Stable Fly as a Carrier of Disease 154
The Role of the Stable Fly in the Transmission of Surra 156

©CB286638

THE

AMERICAN JOURNAL

OF THE MEDICAL SCIENCES

JULY, 1913

ORIGINAL ARTICLES

THE OCCURRENCE OF ANKLE-CLONUS WITHOUT GROSS DISEASE OF THE CENTRAL NERVOUS SYSTEM.

By Wilder Tileston, M.D.,

ASSISTANT PROFESSOR OF MEDICINE, YALE UNIVERSITY MEDICAL SCHOOL, NEW HAVEN, CONNECTICUT.

For a long time ankle-clonus passed as one of the most certain signs of organic disease of the central nervous system, and some authors even asserted that it did not occur in the absence of such disease. In 1879, however, Strümpell[1] pointed out that clonus occurs with considerable frequency in consumptives, in severe cases of typhoid, and in emaciated, weak patients, without other signs pointing to disease of the nervous system, excepting hyperesthesia of the skin and muscles.

It is the purpose of the present article to point out what conditions may be accompanied by ankle-clonus without other evidence suggestive of disease of the nervous system, to present a few illustrative cases, including one in which an autopsy revealed slight microscopic changes in the medulla oblongata, and to discuss the importance of clonus in diagnosis and prognosis. This is desirable for the reason that the subject is not fully treated in the text-books of medicine and neurology.

The Theory of Ankle-clonus. By ankle-clonus in this article is understood the true ankle-clonus, in which, on continuous upward pressure being made on the sole of the foot, there is a series of rhythmical oscillations in plantar flexion and extension, occurring with a frequency of from five to seven in the second, and continuing for a considerable period, often in fact so long as the pressure is maintained. Cases of so-called false clonus, in which a few irregular

[1] Zur Kenntniss d. Sehnenreflexe, Deutsch. Arch. f. klin. Med., 1879, xxiv, 188.

contractions take place, are not included. True ankle-clonus is never found in normal persons, and when present is usually associated with exaggeration of the tendon reflexes. It is rarely present in cases showing increased tendon reflexes due to functional conditions (neurasthenia, etc.), but is frequently observed in organic disease of the cord or brain, on which account it has been assigned considerable diagnostic importance. Clonus is also found, but less frequently, in other situations, especially the knee and jaw. In animals a closely analogous phenomenon is encountered in the so-called scratch reflex, so thoroughly studied by Sherrington.[2] Here in response to a continuous electric stimulation of the skin a series of rhythmical movements of the leg takes place, having a frequency of four in a second. Absolute regularity of the contractions is present in both conditions, and has been shown in the case of the scratch reflex to be due to the presence of a refractory phase, during which stimulation fails to elicit a muscular contraction. The path of the reflex may be assumed to be from the sensory fibers in the gastrocnemius muscle to the lumbar cord, and thence back again to the muscle by its motor nerve. But the reflex is of course more complicated than this, involving coincident inhibition of the antagonistic muscles, and a later contraction of these muscles to bring the foot back to the original position. The close analogy with the scratch reflex, which has been shown to be a spinal reflex, makes it probable that the cerebral cortex is not directly concerned in the production of clonus.

Ankle-clonus in Infectious Diseases Without Gross Lesions of the Central Nervous System. In pulmonary tuberculosis, Strümpell, as already stated, was the first to note the presence of ankle-clonus. He found it of fairly frequent occurrence, especially in emaciated subjects. Longard[3] seems to be the only writer to have made an extensive study of the subject, finding it present in 30 of 82 tuberculous cases, but in only 7 of these was clonus associated with a general increase of the tendon reflexes. Six of these 7 were emaciated and febrile, the other was neither. In 8 cases clonus was noticed in well-nourished patients. He makes no mention of the stage of lung involvement, but from the fact that his cases were observed in a general hospital it may be assumed that they were in an advanced stage. Personally I have not met with clonus in incipient tuberculosis, but only in cases in the last stage.

In typhoid fever the reflexes have been more thoroughly investigated, especially by the French. Remlinger[4] found ankle-clonus in 21 out of 100 cases, but in some of these the clonus was only "suggested." In these 21 cases with clonus the tendon reflexes were increased in 16, normal in 2, diminished in 2, and absent in 1.

[2] The Integrative Action of the Nervous System, London, 1906.
[3] Deutsch. Zeitschr. f. Nervenheilk., 1891, i, 300.
[4] Rev. de Méd., 1901, xxi, 46.

The plantar reflexes were normal in all. The period of the disease in which clonus was first noted was variable, the phenomenon occurring with about equal frequency in the early stage, at the height of the fever, and during defervescence. It is important to note that clonus was more frequent in the severe cases, indicating a high grade of toxemia, for the mortality in the cases with clonus was 28.6 per cent., almost three times that of the cases without clonus (10 per cent.).

In other infectious diseases, such as septicemia (Case I of this paper), pneumonia, dysentery, cholera, malignant syphilis, the occasional presence of clonus has been noted, but no systematic studies have been made.

The following cases observed by the writer will serve to illustrate the occurrence of clonus in infectious diseases. The first is of particular interest because it is one of the few in which a postmortem examination of the central nervous system has been made.

CASE I.—*Septicemia following sinus thrombosis; marked clonus of ankle and jaw; autopsy; no meningitis nor gross lesions of central nervous system; moderate degenerative changes in pons and small foci of myelitis not involving pyramidal tracts.*

F. E., a female, aged fourteen years, entered the Massachusetts Charitable Eye and Ear Infirmary March 28, 1909, suffering from mastoiditis and sinus thrombosis, for which an operation was performed. There followed an intermittent fever, which continued until the death of the patient, April 15. When seen by the writer, April 12, there was marked emaciation, the head was retracted and stiff, and Kernig's sign was present. There was a marked and continuous clonus of the jaw and of both ankles, and the knee-jerks were increased. The plantar reflexes were normal, and there was no spasticity of the legs. Lumbar puncture on two occasions yielded a clear fluid of normal pressure and sterile on cultures. On the fourteenth day the patient had fallen into a condition of stupor. The jaw clonus was less marked, and the ankle-clonus was present on the right side, but only suggested on the left. She died April 15. At the autopsy (Dr. Verhoeff) there was found suppurative mastoiditis, with thrombosis of the lateral sinus at the site of operation, and intense venous congestion and edema of the cerebral pia-arachnoid, but no signs of meningitis; the spinal cord was not examined. There were septic endocarditis of the mitral valve and septic infarcts in the lungs, spleen, and kidneys. There was pus in both pleural cavities. From the spleen the streptococcus was obtained in pure culture.

Microscopic Examination: Unfortunately only the medulla oblongata was available for microscopic study. For the notes the writer is indebted to Prof. E. E. Southard, of the Harvard Medical School: "The positive findings were two in number: (1) A generalized diffuse blackening of the fibers in the white matter

wherever examined, of a sort to suggest curable toxic changes rather than incurably destructive degeneration, and (2) foci of exudative myelitis, in general of an exceedingly mild character, but in one instance striking and associated with alterations in the nerve tissue itself.

"To begin with the focal lesion, we find the meshes of the sheath of the posterior median artery in the posterior median septum densely filled with mononuclear cells, including a few pigment-bearing cells, a small but somewhat larger number of endothelial cells containing mononuclear cells, great numbers of lymphocytes, and a number of plasma cells. The endothelium of the posterior median artery had been largely stripped away and lost. One other small artery in the lateral segment of the bulb was found invested with a slender amount of exudate. The others, including the meningeal arteries were quite free from exudate.

"There is no evidence of spread of exudate into the neighboring bulb tissue. The nerve cells of both nuclei of Goll do not stain properly, but neither do any other nerve cells seen elsewhere look as nerve cells should."

CASE II.—*Severe typhoid fever in a girl, aged eight years; massive hemorrhages on twenty-fifth day, followed by marked ankle-clonus; direct transfusion of blood, with prompt improvement, and disappearance of ankle-clonus in five days.*

B. C., a female child, aged eight years, entered the New Haven Hospital September 7, 1910 (service of Dr. C. J. Bartlett), with typhoid fever. At entrance the knee-jerks were normal. The disease ran a severe course, and on September 25, the twenty-fifth day of the disease, there were two profuse hemorrhages from the bowels. When seen by the writer the next day the child appeared exsanguinated, restless, dyspneic, and thirsty, but was conscious. There was marked and continuous ankle-clonus on both sides, the Achilles and patellar reflexes were greatly increased, but the Babinski and Oppenheim phenomena were both negative and the abdominal reflexes were normal. There was no spasticity. There were no signs of meningitis nor of other disease of the central nervous system. The same day the operation of direct transfusion of blood was performed, with immediate improvement in the alarming condition. The clonus persisted until October 1, when it disappeared and remained absent thereafter. Recovery was uneventful and complete.

CASE III.—*Girl, aged fifteen years; in last stages of phthisis; ankle-clonus five days before death.*

E. K., Russian Jewess, entered the Channing Home for Consumptives October 4, 1903, and died October 9, 1903. Duration of symptoms four months. Physical examination showed an emaciated girl, too weak to sit up. There were signs of extensive consolidation and a cavity on the right side, and of less extensive

involvement of the left lung. There was a typical ankle-clonus on the right side and a slight one on the left. The knee-jerks were increased; the sensation was normal.

CASE IV.—*Girl, aged seventeen years; in third stage of phthisis; ankle-clonus six weeks before death.*

A. E. F., an Irish girl, entered the Channing Home for Consumptives August 31, 1903, with symptoms of three months' duration, and died November 29, 1903. She had lost fourteen pounds in weight, but was still fairly nourished. Temperature, 102°, pulse, 140. The process advanced rapidly, and in a month after admission there was complete consolidation of the right lung and of the left upper lobe. Tubercle bacilli were found in the sputum. Ankle-clonus was found on both sides, with marked exaggeration of the knee-jerks. Four days later the clonus was well marked on the right side, but only slight on the left.

Cases like the foregoing are not uncommon, but heretofore have not been generally recognized. It will be noticed that the clonus may be either one-sided or bilateral, and does not usually persist for more than a few days or weeks. In the writer's experience clonus has been more frequent in the young and in the female sex, but this may be due to the nature of the clinical material.

Ankle-clonus in Cachexia. Sternberg[5] mentions clonus in weak, emaciated patients afflicted with malignant disease and "senile marasmus."

Ankle-clonus in Various Intoxications. In uremia ankle-clonus is not uncommon, and, as Curschmann[6] pointed out, may in chronic cases, come on several days before the uremic seizure, thus furnishing a valuable prognostic indication. Lion[7] also found a marked increase in the tendon reflexes, and sometimes ankle-clonus shortly before the uremic outbreak, though a moderate increase was not uncommon in chronic nephritis in the absence of uremic symptoms. With improvement or recovery he found a return of the reflexes to normal.

During narcosis from ether and chloroform, Goldflam[8] found ankle-clonus frequently. Strümpell and Sternberg mention increased tendon reflexes in acute strychnine poisoning, but do not state whether clonus was present. Lewandowsky[9] states that Syllaba and Crocq noted clonus in chronic mercurial poisoning, but the writer was unable to find a reference to the original article. While chronic alcoholism increases the tendon reflexes it does not apparently give rise to clonus in the absence of organic changes.

An interesting state of the reflexes is found after the use of

[5] Die Sehnenreflexe u. ihre Bedeutung f. d. Pathologie des Nervensystems, Leipzig, 1893, pp. 95 to 97.

[6] Verhandl. d. Kongress f. innere Med., 1909, xxvi, 341.

[7] Zeitschr. z. klin. Med., 1903, l, 257.

[8] Neurol. Centralblatt, 1903, xxii, 1109, 1137.

[9] Handb. d. Neurologie, 1910, i², 603.

hyoscine (scopolamine) in medicinal doses. Ankle-clonus was noted in from one-quarter to one-half the cases by Hahn,[10] Kutner,[11] and Link,[12] and more remarkable still there was a positive Babinski toe sign in a still greater proportion, up to 86 per cent. The Babinski sign appeared in about one-half hour after administration, and after a minimum dose of 0.0004 gm. This production of a positive toe sign indicates a curious selective action on the part of hyoscine not to be found after the use of any other drug, and is one of the few exceptions to the rule that a positive Babinski, when found in adults, indicates organic nervous disease. The tendon reflexes after hyoscine showed no consistent changes, and the muscle tonus was said to be diminished.

Ankle-clonus in Neuroses and Pyschoses. In epilepsy, according to Gowers,[13] ankle-clonus is often to be found immediately after the convulsion, but only for a short time, and Babinski[14] has found his toe sign usually positive at this time.

The occurrence of ankle-clonus in neurasthenia is denied by many authorities, but Oppenheim[15] in his text-book states that it may be found, though rarely. Its presence should make one dissatisfied with the diagnosis of neurasthenia, for in most such cases some organic disease will be found. An instance in point is a case originally reported by Ballet[15] as one of neurasthenia, with ankle-clonus, but shown by the autopsy some years later to have been syphilitic meningitis.

In the case of hysteria there is also much difference of opinion. Most authorities are agreed that ankle-clonus does not occur in hysteria unless there is paralysis, and that it is absent in the common flaccid type of paralysis. In the hysteric spastic paraplegia, with contracture, however, ankle-clonus has been described by many authors, chiefly of the German school. Oppenheim and von Monakow[17] consider it rare, while Strümpell[18] says it is often encountered in this form of paralysis. Babinski[19] claims that when clonus is met with in hysteria it is always due to some extraneous factor, such as organic disease or excessive fatigue. The presence of ankle-clonus in hysteric subjects should therefore always arouse a suspicion of organic nervous disease, which becomes a certainty if the Babinski toe sign is present, for this is never met with in uncomplicated hysteria.

[10] Neurol. Centralblatt, 1911, xxx, 114, 185.
[11] Deutsch. med. Woch., 1907, xxxiii, 98.
[12] Zeitschr. f. klin. Med., 1906, lix, 252.
[13] Epilepsy and Other Chronic Convulsive Diseases, London, 1901, 2d edit.
[14] Rev. neurologique, 1899, vii, 512.
[15] Lehrbuch d. Nervenkrankheiten, V Auflage, 1908.
[16] Rev. neurologique, 1903, xi, 234; 1905, xiii, 732.
[17] Gehirnpathologie, II Auflage, 1905, p. 489.
[18] Lehrbuch d. spec. Path. u. Therap., XIII Auflage, Band iii, S. 615.
[19] L'Encéphale, Paris, 1909, iv[1], 40.

In paralysis agitans, according to Oppenheim, the tremor of the foot may simulate clonus, and very rarely a true clonus may be present.

Bonhoeffer[20] and Westphal[21] have found transitory ankle-clonus, with spastic gait, in acute psychoses in the stage of excitement and loss of weight; Bonhoeffer speaks of his cases as psychosis with anxiety ("Angstpsychosen"), without giving a more definite diagnosis.

Ankle-clonus in Arthritis. It is important to remember that chronic disease of a joint often causes increased reflexes of the corresponding limb, and if the ankle is involved, ankle-clonus is by no means uncommon, apart from any organic nervous disease. In acute articular rheumatism clonus has not been reported, probably owing to the immobilization of the joints by muscle spasm. Jones[22] mentions the presence of jaw-clonus when the articulations of the jaw are involved. Clonus may occur in various types of chronic arthritis. Thus the writer has seen it in the gonococcal and chronic infectious types, while Jones emphasizes it in rheumatoid arthritis. The mechanism of its production is not clear, but might be explained on the ground that frequent painful stimuli from the joint put the corrresponding segment of the cord into a state of overexcitability, so that the stimulation of the Achilles tendon results in clonus.

Ankle-clonus after Fatigue. Several authors state that ankle-clonus may be found after unusual fatigue, or loss of sleep combined with anxiety, and Auerbach[23] found clonus in 2 out of 39 bicyclists after long distance (50 to 100 km.) races. After long-distance runs, however, the results are different, Knapp and Thomas[24] and Oeconomakis[25] finding no instances of clonus, but rather a diminution of the tendon reflexes, after "Marathon" races of 42 km. The difference may be due, as Oeconomakis suggests, to the fact that the calf muscles are comparatively little used in bicycling, while in running there is a chance for the reflex arc to become exhausted.

Graphic Studies of Ankle-clonus. The most extensive investigation of clonus by graphic methods has been made by Levi.[26] He found that the clonus associated with organic disease showed in the tracing oscillations of equal height and equally spaced, while in the pseudoclonus found in functional diseases and in convalescence from acute infections (only one case studied) the oscillations were of unequal height, giving an irregular appearance to the

[20] Seitenstrangerscheinungen bei akuten Psychosen. Psychiatrische Abhandlungen herausgeg. v. C. Wernicke, Breslau, 1896, Heft ii, p. 1.

[21] Neurolog. Centralbl., 1903, xxii, 12 (foot-note).

[22] Lancet, 1902, ii, 1746.

[23] Neurolog. Centralbl., 1905, xxiv, 251.

[24] Jour. Nerv. and Ment. Dis., 1904, xxxi, 94.

[25] Neurol. Centralbl., 1907, xxvi, 498, 563.

[26] Arbeiten. a. d. Neurolog. Institut. a. d. Wiener Universität, 1907, xvi², 27.

curve. In one of his cases, diagnosticated as hysteroneurasthenia, the clonus to the naked eye seemed to be genuine, and only the tracing proved it to be "false." Levi claims that true clonus is always a sign of organic disease, provided that the graphic method has been employed. The string galvanometer of Einthoven has also been used by Salomonson[27] in the study of clonus, and he, too, found a perfectly regular curve in the case of organic disease, while hysteria showed, besides the action currents of the clonus, those of voluntary muscle tetanus. These methods appear to offer a valuable way of studying clonus, but the observations have not been extended enough to be conclusive. It is desirable that further studies with them should be made in cases of ankle-clonus without organic disease of the nervous system.

The State of the Skin and Tendon Reflexes. In the class of cases under consideration the skin reflexes may be increased or diminished, but Babinski's toe sign is almost invariably absent, an important distinction from clonus due to organic disease, where it is usually to be elicited. The sole exceptions to this rule are clonus after the use of hyoscine and immediately after the epileptic convulsion, in both of which conditions a positive Babinski is the rule. The same holds true of Oppenheim's sign. The tendon reflexes are usually but not always increased, when clonus is present, whether it be due to organic or functional disease.

Pathological Anatomy of Clonus Unassociated With Organic Disease. Up to the present but three cases have been studied anatomically, viz., those of Strümpell,[28] Sternberg,[29] and the writer's first case. Strümpell's case was one dying of phthisis, and the spinal cord was found normal, both macroscopically and microscopically. This, however, was in 1873, before the introduction of the finer stains for nervous tissue. Sternberg's case was also one of phthisis, and the spinal cord is said to have been normal on microscopic examination, but no details are given. In the writer's case foci of myelitis were found in the posterior columns in the bulb, as well as diffuse Marchi degenerations. Not too much stress should be laid on the latter, however, for Gay and Southard[30] found such diffuse changes in a considerable proportion of cases dying of acute terminal infections. Further studies, including both brain and spinal cord, are necessary before the question of the absence of "organic" changes can be settled.

Explanation of the Occurrence of Clonus in the Absence of Organic Nervous Changes. It will be remarked that practically all the conditions under discussion are accompanied by toxemia, usually of a serious nature. Even in the case of clonus after excessive fatigue there is reason to believe that toxic products are at work. It is possible that the deleterious substances affect only the function

[27] Folia neuro-biol., 1910, iv, 1.
[28] Loc. cit.
[29] Loc. cit.
[30] Centralbl. f. Bakt., Parasitenkunde u. Infectionskrank., 1910, lv, 117.

of the nervous tissue, but it is also not improbable that there are structural changes, such as Edinger,[31] for instance, has shown to take place in rats after overexertion. Such changes, however, must be capable of complete recovery, for the clonus may disappear quickly, not to return. We are still in doubt as to the exact way in which clonus is produced. If, with Oppenheim, we regard ankle-clonus simply as an exaggeration of the Achilles reflex, we might suppose that the toxic substances rendered the nerve fibers, or perhaps the synapse in the cord, more irritable. The phenomenon cannot be accounted for solely by the removal of cortical inhibition by reason of a lesion of the pyramidal tracts, for clonus is sometimes found in cases of organic disease where these tracts are uninjured, and it may be lacking where they are completely degenerated. The bulbar lesions in the writer's first case are suggestive of the possibility that bulbospinal or bulbothalamal tracts might be of importance in the production of ankle-clonus.

Diagnostic Considerations. The fact that ankle-clonus may appear in so many and varied conditions of toxemia, quite apart from any demonstrable lesion of the central nervous system, makes it necessary to restrict the diagnostic importance heretofore attached to this sign. In the absence of these toxic states, however, it has still a great deal of practical value in the diagnosis of organic nervous disease. The probability of such disease is greatly enhanced if there is in addition to clonus a positive Babinski or Oppenheim sign, as these do not occur in the toxic conditions under discussion, with the exception of hyoscine intoxication and the epileptic attack. The presence of a positive Mendel sign (plantar flexion of the toes on percussing the outer border of the foot over the fifth metatarsal bone), although considerably rarer than the other toe phenomena, appears to be conclusive evidence of organic disease. The complete absence of spasticity and of other signs pointing to organic lesions is also of importance in distinguishing the "functional," or "toxic" cases. On account of the rarity of true clonus in uncomplicated neurasthenia and hysteria its presence should always arouse the suspicion of coëxisting organic disease of the nervous system.

Prognostic Value. In the infectious diseases the appearance of ankle-clonus certainly adds to the gravity of the prognosis, as it indicates a high degree of toxemia, but it does not preclude the possibility of recovery. In nephritis it may be an important sign of the approaching uremic outbreak. The disappearance of the clonus is a favorable sign, except in the severest cases, where it may be only an indication of impending death.

1. Summary and Conclusions. Ankle-clonus indistinguishable from the genuine may be found more or less frequently in a variety of conditions, without accompanying organic nervous disease.

[31] Verhandl d. Kongress f. inn. Med., 1898, xvi, 292.

2. These conditions are: (*a*) Acute infectious diseases, especially typhoid; (*b*) chronic infections associated with marked toxemia, especially tuberculosis of the lungs in the third stage; (*c*) uremia shortly before and during the acute uremic seizure; (*d*) epilepsy immediately after the convulsion; (*e*) intoxication from certain drugs, *e. g.*, hyoscine, ether, and chloroform; (*f*) excessive fatigue; (*g*) exceptional cases of certain neuroses, viz., neurasthenia, hysteria, paralysis agitans; (*h*) psychoses in the stage of excitement; (*i*) chronic articular rheumatism.

3. With the exception of joint disease, a toxic action on the nervous system may be assumed in all these states as the underlying factor in the production of clonus. This is obvious in the case of the infectious diseases and drug intoxications; in uremia, epilepsy, and undue fatigue the presence of toxic products of metabolism may be regarded as probable, though not yet demonstrated, and even in hysteria and neurasthenia the action of toxic products in severe cases cannot be excluded.

4. In the case of articular rheumatism a constant spinal irritation from the inflamed joint tissues is the probable cause.

5. In two autopsies on cases of phthisis, with clonus, no changes were found in the central nervous system. In the writer's case, however, inflammatory exudate was demonstrated about the posterior median artery in the posterior septum of the bulb.

6. Clonus due to toxic states usually may be distinguished from that of organic nervous disease by the absence of spasticity and of other signs pointing to organic disease, and particularly by the absence of the Babinski and Oppenheim toe signs.

7. An exception to the above rule is encountered after the use of hyoscine in medicinal doses and immediately after the epileptic attack, in both of which instances the Babinski and Oppenheim signs may be positive.

8. The occurrence of ankle-clonus is of prognostic value in uremia, preceding at times the acute seizure. Ankle-clonus usually disappears a few days before death, otherwise its disappearance usually indicates an improvement in the patient's condition.

THE RATIONAL TREATMENT OF SURGICAL OR NON-PULMONARY TUBERCULOSIS.

By John B. Hawes, 2d, M.D.,

DIRECTOR, TUBERCULIN DEPARTMENT, ASSISTANT VISITING PHYSICIAN, MASSACHUSETTS GENERAL HOSPITAL; SECRETARY, BOARD OF TRUSTEES MASSACHUSETTS HOSPITALS FOR CONSUMPTIVES.

I do not intend this article to be an argument against the proper use of surgery in surgical tuberculosis. I shall endeavor to show,

however, that in the majority of cases of non-pulmonary tuberculosis surgical interference is but an incident in a course of treatment the most important part of which comes before and after the operation. In other words, in practically no case of surgical tuberculosis can the operator, no matter how skilful, remove all traces of tuberculosis; in tuberculous cervical adenitis, for instance, it is not uncommon to meet patients with large disfiguring scars in the neck and with a history of recurrence of the glands soon after the operation. These patients are recorded on the hospital records as "discharged cured." Only too often subsequent events demonstrate the fallacy of this statement.

During the past three or four years the attitude of many surgeons has undergone a radical change as to the treatment of surgical tuberculosis. While there still remain certain cases in which extensive surgical procedures are indicated, such as in renal tuberculosis, surgeons are coming to realize more and more that their duty to the patient does not end with the completion of the operation and the discharge of the patient from the hospital; they are recognizing the fact that only by constant careful supervision for many months afterward can a permanent cure be obtained. Further than this they are learning that equally careful and constant supervision prior to any surgical procedure will vastly increase the benefits of any operation and in many cases do away with the need of it altogether. This paper is a description of the practical application of these principles as carried out in my clinic at the Massachusetts General Hospital.

In July, 1905, Dr. Joseph H. Pratt, of Boston, started the first "tuberculosis class" in this country. This class consisted of a selected group of consumptive patients who were taught by means of weekly meetings and home supervision how to carry on what was known as "home sanatorium treatment." At the start I became deeply interested in this movement, and soon formed such a class among my own patients. As time went on the number of beds for indigent consumptives in Massachusetts greatly increased, and thus the sphere of usefulness for tuberculosis classes became more and more limited until at the present time in this State one can assign to the tuberculosis class only a small part in the campaign against pulmonary tuberculosis.

This is not the case with surgical tuberculosis, however, There are few beds for children and practically no beds for adults in sanatoria or hospitals in this State where patients with non-pulmonary surgical tuberculosis can receive adequate outdoor hygienic sanatorium treatment of which they are so badly in need. The reasons for this are obvious in that surgical tuberculosis is rarely a danger to the community which has its hands full in caring for its dangerous and infectious cases of consumption. These reasons, though perfectly adequate, render the case of the patient so un-

fortunate as to have tuberculosis of glands, kidney, etc., instead of his lungs a very deplorable one. It became increasingly evident to me that while the usefulness of the home sanatorium movement must become less and less as far as consumption of the lungs was concerned, in surgical tuberculosis it was and will continue to be a potent agent for good.

The hospital authorities have seen fit to call this department which I have built up and devoted to the care of surgical tuberculosis a "tuberculin clinic." This is a misleading name. It is true that with the great majority of my patients I use tuberculin in conjunction with other measures, but in only a few cases would I be willing to attribute to tuberculin all or nearly all of the improvement which has usually taken place. In a certain class of patients with tuberculosis of the genito-urinary tract it has seemed to me that tuberculin is the most important factor in treatment; in the great majority of cases, however, it is undoubtedly a factor, but by no means the most important factor in producing good results. From the start I have confined myself to the use of a bouillon filtrate tuberculin kindly supplied me by Dr. E. R. Baldwin, of the Saranac Lake Laboratory. Other investigators have advocated the bacillary emulsion and certain other preparations, notably bovine tuberculin. My results with the B. F. preparation have been so satisfactory that I see no reason for a change.

Tuberculin is administered once a week according to the well-known rules laid down by Trudeau. The initial dose is 0.0001 to 0.0005 mg., rarely 0.001 mg. This is gradually increased up to 50 to 100 mg. Increase of dosage is gauged by careful observation of clinical signs of a reaction—local, focal, or constitutional. A few patients take their pulse and temperature regularly at home; in the majority of instances this is unnecessary. Constitutional reactions have been exceedingly rare, and in no case has the slightest harm resulted from such occasional reactions while in not a few instances marked improvement seems to have followed a mild constitutional disturbance. It has been my aim to avoid all constitutional reaction by repeating the same dose or decreasing it. I have found it possible to carry most of my patients up to large amounts of tuberculin without the slightest discomfort or disability.

The clinic is held once a week at the out-patient department of the Massachusetts General Hospital; the hospital provides me with a room, necessary equipment, and a nurse. Patients are referred to me from the other out-patient departments, particularly the male and female surgical and genito-urinary departments. Upon the arrival of any new patient, after a physical examination to rule out any pulmonary condition, a card is filled out recording temperature, pulse, and weight. The methods of treatment and reasons for everything that is asked are explained in detail; the patient is then sent to the Social Service Department (without which or its equivalent no such clinic could exist) with the request:

"Please investigate and report to me as to home conditions." This is followed by a personal interview with the social worker taking up the case, in which the exact needs of this individual patient are explained. The chief of the department from whom the patient was referred is also seen and his ideas obtained as to treatment of the local condition. In every case of tuberculous adenitis the patient is sent to the dental department for thorough cleaning of the teeth and to the throat department for tonsillectomy or other procedure if this is deemed advisable. All this takes time. It is often one or two weeks before all these important details can be attended to. At the outset every effort is made to secure the patient's coöperation and to see that he or she understands the reason for everything.

Patients begin to arrive shortly before 9 A.M. Men, women, and children are seen together in one large room. Each patient, on arrival, is given a numbered slip; temperature, pulse, and weight are recorded on the history cards, which are arranged in order at the table where I see the patients. One after another as I call out their names the patients come and talk over with me the details of the past week. If the week has been an uneventful one, and the patient has had no signs of a reaction, constitutional or local, the interview is short; on the other hand, if the patient is not doing well, has lost weight, or is not following out directions it may take some time. Most of the patients I know well and call by their first names; they come to know each other and to enjoy the weekly meetings. I often call the attention of the entire group to striking points coming up in the course of my conversation with individual patients. Frequent consultations are held with the physicians and surgeons of other departments who are looking after the local conditions. The lungs are carefully watched, and whenever the slightest suspicion of pulmonary involvement is found radical treatment usually in a sanatorium is instituted.

Work vs. Rest. Many of these patients keep at their regular work during the entire course of active treatment. While it is advisable and necessary in the majority of instances that all work or school be given up at least for the first two or three months, in not a small number the general condition has been such that it has seemed a useless hardship to impose enforced idleness.

Outdoor Sleeping. During the summer months many patients sleep outdoors; a few sleep out all the year round. The question of fresh air at night is carefully gone into in every case; and in many instances patients are allowed to go to school or to work on condition that they get a sufficient amount of fresh air at night.

Length of Stay in the Clinic. Patients are urged to attend regularly once a week until the process is cured or arrested. This cannot be done in every case, and, as a general rule, a compromise has to be made. As the local process and general condition improve patients are frequently allowed to come once in two weeks instead

of every week. When the disease is apparently arrested they report once a month or once in two months until a permanent cure is assured.

Class of Cases. The greater number of patients in this clinic are those with tuberculous cervical adenitis; in addition to this there are many cases of genito-urinary tuberculosis, tuberculosis of epididymis, prostate, bladder, tubes, ureter, or kidneys. Other less common cases are those with bone or joint disease, lupus, ocular, and mesenteric gland tuberculosis.

Diet and Drugs. If the patient is under weight one quart of milk a day in addition to the usual three meals is prescribed; occasionally olive oil in tablespoonful doses after meals is ordered. Unless the patients are distinctly in need of more nourishment nothing extra in the way of food is ordered, providing that it is definitely ascertained that three really good meals a day are assured. Drugs are rarely if ever used.

Statistics and Results. Since the time that this clinic has been devoted purely to extrapulmonary tuberculosis I have had 209 patients under treatment and observation. Of these 209 patients 50 are in regular attendance every week or every other week, and are therefore not included; 43 patients for one reason or another remained under treatment too short a time to allow of their being considered in this series. The chief causes of this too short a stay at the clinic were:

(*a*) The presence of pulmonary tuberculosis which necessitated removal to a sanatorium as soon as possible.

(*b*) The patient's home was too far away to allow of regular attendance.

(*c*) The home and financial situation were such that the patient was either unwilling or unable to attend regularly.

This leaves 116 patients of whom I can speak with considerable certainty as to results. Each of these has been under more or less constant supervision, coming to report once a month or once in two or three months; the minimum period since attending regularly has been six months; in a large proportion of cases it has been two to four years since they were regular members of this department. These cases are divided as follows:

Tuberculous adenitis	60
Ocular tuberculosis	28
Tuberculosis of kidney	8
Tuberculosis of epididymis	5
Tuberculous tenosynovitis	3
Bone tuberculosis	3
Lupus	3
Mesenteric gland tuberculosis	2
Tuberculous peritonitis	1
Tuberculous salpingitis	1
Tuberculous prostate	1
Tuberculous fistula in ano	1
	116

I have used the terms adopted by the National Tuberculosis Association to denote results. This omits the term "apparently cured" substituting the more conservative one "disease arrested." Of the 60 cases of tuberculous adenitis, which includes young and old patients, those with merely one small broken-down gland and those with extensive bilateral processes on which many operations had previously been performed with no permanent benefit, 46 have had the disease arrested and are now well and healthy in every way, 13 have been markedly improved, while in only 2 instances has the disease progressed despite treatment. There have been no deaths. Of the 28 patients with ocular tuberculosis in some form 9 have had the disease arrested, 17 have been markedly improved, while in 2 cases the process has advanced. Of the 28 remaining cases, which include various other forms of non-pulmonary tuberculosis, 14 have had their disease arrested, 12 have improved, while in 2 the disease has progressed.

These results are by no means startling, although on the whole they are eminently satisfactory when one considers (1) the absolute lack of sanatorium facilities, (2) the comparative lack of adequate home supervision, and (3) the financial condition of most of these patients, which makes it necessary for by far the greater number of the adults to get to work much sooner than would have been the case under ideal conditions.

As stated in the beginning of this paper, I do not attribute to tuberculin alone all or nearly all of whatever improvement has been brought about. It is a judicious combination of proper hygiene, conservative surgery, and tuberculin backed up by individualization in each case and the employment of applied commonsense that has helped these patients in the past and that will help similar patients in the future.

MYOCARDIAL HYDROTHORAX, WITH REPORTS OF CASES.[1]

By James M. Anders, M.D., LL.D.,
PROFESSOR OF MEDICINE IN THE MEDICO-CHIRURGICAL COLLEGE OF PHILADELPHIA.

A collection of transuded serum in the pleural cavities is a secondary condition, most commonly associated with either cardiac or renal dropsy or the severe anemias, for example, leukemia and progressive pernicious anemia. Not infrequently, hydrothorax also supervenes in chronic dysentery, chronic diarrhea, carcinoma, syphilis, and scurvy. In all of the chronic diseases mentioned

[1] Read before a stated meeting of the College of Physicians of Philadelphia, February 5, 1913.

the condition is in most cases, at least, a late development, often in the course of general dropsy, but such familiar examples are not included in the present discussion. It is also my purpose to exclude from consideration in this paper, hydrothorax dependent upon local stasis due to new-growths of the pleura, lungs, and diaphragm.

The renal group is a large one, and while the cases of hydrothorax that are secondary to chronic parenchymatous nephritis are not considered here, those that come on in the course of chronic interstitial nephritis are unilateral, as a rule, for longer or shorter periods of time, are caused by foregoing dilatation of the heart, and are included with the purely myocardial form. In the latter class both heart and kidney conditions are due to one and the same cause, namely, arteriosclerosis. It is readily conceded that in valvular heart affections attended with general venous engorgement the process may be unilateral for a time in consequence of local obstruction, but this group of cases is also eliminated from full consideration here, although reference will be made hereafter to its relative frequency as compared with myocardial hydrothorax. In short, this paper deals solely with instances of hydrothorax due to myocardial disease, but with some of which chronic interstitial or atrophic nephritis was associated.

The opinion commonly prevails among writers that in cachectic and renal cases, hydrothorax is usually bilateral. But though transudation is, in the majority of cases, bilateral, certain American authors, (Stengel, Osler, and Pepper) have insisted upon the view that it is sometimes unilateral throughout the whole or greater part of its course, for example, in the cardiac group of cases, which may be occasioned either by myocardial changes or chronic valvulitis, and when so is usually right-sided and may be unaccompanied by external dropsy.

Causes of Myocardial Hydrothorax. The association of cardiac disease and unilateral hydrothorax is not rare. This view is confirmed by the observations of Lord,[2] who found in a series of fifteen cases that six were unilateral and right-sided, of which five were complicated with heart lesions. It is a point of major significance that in myocardial disease the same mechanical influences are at work to bring about a pleural transudate as in valvular disease, principally cardiac dilatation, and more particularly of the right auricle. Moreover, the foregoing fact enables us to understand the reason for the election of the right side of the thorax as the seat of the serous transudate, in cases in which the condition originates primarily in sclerosis of the bloodvessels. Toxic poisoning of the myocardium or of the nerve centres is most probably also a causative factor of first-rate importance in the production of myocardial hydrothorax.

[2] Osler's Modern Medicine, iv, 848

Again, cases of apparently incipient arteriosclerosis, so far as the peripheral bloodvessels are concerned, may show more marked changes in the small arteries that supply the viscera, the heart, and kidneys inclusive. This has been revealed by recent pathologic studies, and in such instances both the cardiac insufficiency on which the hydrothorax depends and also the urinary phenomena when present are produced by a more or less hidden arteriosclerosis.

On reviewing the literature bearing upon the immediate causes of the cardiac variety of pleural transudate one finds that several recent theories have been advanced and ably championed. A considerable number of writers, among them Cardanelli, Villani, Howe, Lizzatto, and Pepper, attributed the occurrence of right-sided transudation of serum to an old pleural inflammation of low grade, which had so altered the perivascular tissue "that subsequently serous extravasation occurred with general venous stasis that had not become sufficient to cause external or bilateral pleural edema." Stengel[3] cites Villani and Peter as suggesting that irritations or inflammations of the liver may extend through the diaphragm to the pleura and thus occasion a combined pleural exudate and transudate.

German writers have also called attention to this variety of the condition in many forms of liver and heart disease, including chronic myocarditis. These may show marked latency so far as general symptoms are concerned, to be followed after a variable length of time by edema of the legs, hepatic enlargement, and finally a left-sided transudate.

More recently, competent observers have noted the occurrence not uncommonly of unilateral or right-sided hydrothorax independently of hepatic disease. Jaccoud first suggested pressure upon the azygos vein and superior vena cava as the cause. Stengel, who arrived at the same conclusion independently, argues from anatomic considerations, that is the relationship of the chest vessels to adjacent structures, that "even a moderate dilatation of the right auricle and cavæ must of necessity exercise considerable compression upon the azygos vein and thus reduce its lumen." Of course, similar conditions do not obtain on the left side of the chest.

More recently still, Fetterolf and Landis[4] have, as the result of their investigations, concluded that the serous fluid comes from the visceral and not from either the parietal pleura or azygos veins, the outpouring being caused, "so far as the pressure factor is concerned, by dilated portions of the heart pressing on and partly occluding the pulmonary veins." They continue: "Greater frequency on the right side is due to the fact that dilatation of the

[3] Univ. Penn. Med. Bull., June, 1901.
[4] Amer. Jour. Med. Sci., November, 1909.

right auricle is more common and more easy than a similar condition of the left side, and such dilatation is the only factor needed to cause damming back in the right pulmonary veins. On the left side, in order to include both upper and lower veins, there is needed dilatation of the left auricular appendix and of the left ventricle, with possibly a retrodisplacement of the vertical septum mentioned above, three factors as against one on the right side." In 13 out of my 16 cases of the myocardial form the hydrothorax was wholly on the right side throughout.

Incidence of Myocardial Hydrothorax. Of 27 cases of hydrothorax due to heart lesions that have fallen under my observation, and of which I have clinical notes, not less than 16 (59 per cent.) were apparently caused by myocardial disease. In 5 of the 16 cases clear and convincing indications of well-pronounced chronic interstitial nephritis were coexistent. These were doubtless instances primarily of arteriosclerosis to which both the myocardial changes and the nephritis were secondary.

In 8 cases only slight evidences of arteriosclerosis coexisted, so that the probabilities are that either the myocardial degenerative and inflammatory lesions were primary or the changes in the blood-vessels of the heart and other viscera were decidedly more marked than in those that are accessible. In 9 cases, however (56 per cent.), the cardiac incompetency which led to the production of the hydrothorax was caused by chronic myocarditis, as will appear evident hereafter.

In none of the 16 cases were the clinical evidences of foregoing valvular sclerosis found. Of course, these figures are too small to base thereon safe inferences, and it is well known that individual experiences differ to a marked degree in relation to special conditions, yet there can be no room to doubt that hydrothorax due to chronic myocarditis is more common than has been supposed. In this connection it is interesting to note that all of my cases occurred in males.

Diagnosis of Myocardial Hydrothorax. Cases of myocardial hydrothorax are often characterized by extreme latency, particularly during the earlier portion of their course, the only subjective symptoms complained of being dyspnea especially on unwonted effort, and a dry, unproductive cough, which is not especially annoying. This was true of most instances observed by me, although in all the signs and symptoms of chronic myocarditis, for example, cardiac dilatation, with hypertrophy, commonly hypertension, arrhythmia, dyspnea, and in 5 cases, as before stated, those of chronic interstitial nephritis, were present.

Obviously a careful physical examination of the entire thorax would serve to enlighten the clinician, but is, according to my personal experience, often neglected because the hydrothorax which gives rise in a measure, at least, to the dyspnea and cough

is unsuspected. Moreover, my observation confirms the view that a serous collection of considerable magnitude in the right pleural sac may go unrecognized in the hands of a physician who is not fairly expert in the matter of physical examination. The physical signs are the same as those of pleurisy, with effusion, but, unlike the latter, serous collections are never encapsulated.

Osler[5] points out that postmortem records show how frequently the condition is overlooked. This fact is applicable in a special degree to the cases that arise in the course of chronic myocardial changes after secondary dilatation supervenes, without external edema, and independently of valvular sclerosis. For example, in three of the five cases of hydrothorax which had not been recognized no external edema coexisted until late in the course of the condition.

The principal error in diagnosis then, it has seemed to me, is in the assumption that hydrothorax is not to be expected in cases of cardiac disease in which the signs of chronic valvulitis and external edema are absent. Obviously, not all cases of arteriosclerosis, with hypertension in which dyspnea whether on exertion or otherwise is present, are due to hydrothorax, since this is a striking feature of chronic myocarditis, as a rule, but if on close examination hydrothorax is found to be absent in this condition (chronic myocarditis) it should be recollected that, as pointed out by Janeway,[6] it indicates marked danger of cardiac insufficiency.

Of the 16 instances of myocardial hydrothorax that have fallen under the writer's observation not less than 5 had been unrecognized, the severe dyspnea having been attributed mainly, at least, to so-called cardiac or cardiorenal asthma and pulmonary congestion. The symptoms and physical signs, however, give a characteristic picture, although it is sometimes difficult to differentiate the cause, chronic myocarditis, from other underlying disease states in which hydrothorax supervenes.

It is not uncommon to meet with cases of myocardial insufficiency that simulate closely those of valvular disease, particularly mitral incompetency, with which may be associated evidence of a mild grade of stenosis. There is, however, not obtainable a clear history of acute articular rheumatism in chronic myocarditis, but commonly of one of the exciting factors that may precipitate secondary dilatation of the heart, such as physical or mental overstrain, an intercurrent febrile affection and the like. Moreover, the apical murmur has a more limited area of transmission in nonvalvular cases, and if the heart be "whipped up" by the use of cardiac stimulants it loses in intensity and may even disappear.

It is said that, as a rule, transudation is not excessive, but in

[5] Text-book of Medicine, p. 668.
[6] Jour. Amer. Med. Assoc., December 14, 1912.

two of my cases as much as 2000 c.c. of serum were removed on several occasions, and in one case not less than 6500 c.c. were withdrawn at a single operation. In 15 out of the 16 cases of myocardial hydrothorax, tappings were carried out and no instances of mixed transudate and exudate were met with, although in one case of a right-sided transudate a left sided pleuritis with effusion was associated. While at first sight it may seem trite to suggest that more or less suspicious cases should be needled, there can be little doubt that this simple procedure, which gives us reliable information as to the existence and nature of the process, is not resorted to as regularly as it deserves to be.

Course and Prognosis. While death is inevitable in by far the majority of cases, soon or late, even after marked improvement or apparent comfort has been brought about as the result of treatment, hydrothorax may develop, as my clinical records show at a comparatively early stage of arterial or myocardial disease without recurrence of the condition, if appropriately treated, and life prolonged indefinitely. Thus one of my cases has shown persistent good health for fifteen years following repeated aspirations, and the use of cardiac tonics and stimulants as well as other measures; another for a period of twenty months, and still others for shorter intervals of time.

Treatment. The treatment of this form of hydrothorax must have the same objects in view as in the other varieties, and has reference to the removal of the transudate by tapping the chest, and, so far as possible, of the causative condition by hygienic and medicinal means. It is futile, as a rule, to attempt to get rid of the transudate by the exhibition of digitalis and other cardiac stimulants without first withdrawing the fluid by aspiration if it be considerable in amount. I have repeatedly observed that this class of drugs only tends to aggravate the dyspnea without either diminishing the amount of the transudate or increasing, to an appreciable extent, the urinary secretion.

On the other hand, after removing the fluid by means of thoracentesis, cardiac stimulants often take hold and are of signal service in overcoming the dilatation of the heart and preventing a recurrence of the transudate. The myocardial insufficiency in these cases demands a resort to remedies that will strengthen the heart muscle, for example, digitalis, strophanthus and the like, independently of an increased blood pressure, although nitroglycerin should be combined with these drugs if the arterial tension be decidedly elevated.

Rest is a most valuable adjunct in the treatment of the cardiac dilatation to which the hydrothorax is due; it must be, however, absolute and long continued. The use of saline laxatives carried to the point of rather active catharsis—three or four fluid evacuations daily—proved of decided service in a few of the cases.

In five instances of the series herewith reported a salt-poor diet was employed, with favorable effect, reaccumulation of the transudate being thereby noticeably delayed.

Brief notes of my series of cases are here appended:

CASE I.—Col. M. M. J., aged sixty-two years, occupation, Quartermaster U. S. A., consulted me March 25, 1898.

Family previous and social histories negative. The present illness began in March, 1897, with symptoms of neurasthenia and indigestion. He later developed dyspnea and fatigue on effort, and, as his condition did not improve, he was referred to me by his physician.

At my first examination I found the patient suffering from marked dyspnea, a moderate increase of blood pressure, with accentuation of second aortic sound, a dilated heart, and the physical signs of right-sided hydrothorax, the sac being about one-half filled. The urine contained an occasional trace of albumin and a few narrow hyaline casts; specific gravity, 1010 to 1015.

Aspiration followed by complete rest for a period of two months and the use of cardiac stimulants and a non-nitrogenous diet caused the dyspnea and other symptoms to disappear. At the end of six months he again enjoyed good health, which persisted until one year ago, when he died of an acute illness.

CASE II.—F. L. M., male, aged fifty-seven years; occupation, general shipping agent.

Family History. Negative. The patient was subject to tonsillitis during adolescence; occasional mild attacks of rheumatism. Six years ago he was told by a physician that he had "kidney trouble," was ill for one month. Habits good, excepting excessive use of tobacco.

The present illness began six weeks ago, when the patient noticed dyspnea, palpitation, and loss of weight and strength. When I first saw him on March 17, 1908, there was evidence of marked arteriosclerosis, heart hypertrophied, second aortic sound much accentuated, and a moderate amount of free fluid in the left pleural sac, none in the right, and some edema of the feet and ankles. Examination of the urine: specific gravity, 1010; albumin, a small ring; casts, a few hyaline and finely granular; daily amount, 25 ounces. Blood pressure: systolic, 232 mm. Hg. On May 23 the left chest was aspirated and 17 ounces of fluid, having a specific gravity of 1014, were removed. The dyspnea was much improved. The patient left the hospital three days later and no subsequent report on his condition has been received.

CASE III.—H. L. J., male, aged forty-eight years; occupation, fireman, formerly miner.

Family History. Two brothers, a nephew, and a niece died of tuberculosis, otherwise negative. Habits: excessive use of coffee and alcohol for many years.

The patient first became indisposed on August 27, 1908, when he noticed some shortness of breath on exertion, which rapidly increased. When admitted to the Medico-Chirurgical Hospital on October 17, 1908, he complained severely of dyspnea, pain in the left chest, aggravated by breathing and by coughing; mucopurulent expectoration and occasional attacks of vomiting. The patient was cyanosed and an examination revealed an enormously hypertrophied and dilated heart, with weak, rapid pulse; no free fluid in the chest, though numerous fine moist rales were audible over the bases of the lungs. There was marked edema of the feet and ankles. The urine showed a heavy ring of albumin and a few hyaline and finely granular casts. Systolic blood pressure, 180 mm. Hg. No tubercle bacilli in sputum; numerous staphylococci and streptococci present. On October 26 free fluid was noted in both pleural sacs, dyspnea increasing, while pain in the left chest had ceased. Three days later the left chest was aspirated and 21 ounces of cloudy serum, having the characteristics of an exudate, were removed. Cultural studies revealed Streptococcus pyogenes. The following day the right chest was punctured and 20 ounces of sterile fluid, having a specific gravity of 1014, were withdrawn. Following this operation the hydrothorax showed no tendency to reaccumulate. On the other hand, the left-sided pleurisy persisted and the exudate was removed on two other occasions before the death of the patient, on November 9, 1908.

Summary of the autopsy findings: Heart hypertrophied and much dilated, especially the right auricle; no fluid in right pleural sac which was normal in appearance; extensive left-sided empyema, with recent pleural adhesions; kidneys showed congestion and diffuse nephritis.

Case IV.—A. E., male, aged forty-six years; occupation, police officer.

Family History. Father died of heart and kidney disease. Patient has consumed from 15 to 20 "whiskies" daily for the past eight years.

The present illness began in November, 1908, with gradually increasing shortness of breath and marked edema of the legs. Under treatment the patient improved somewhat for a time, but soon after returning to his occupation there was a recurrence of the previous symptoms. When admitted to the Medico-Chirurgical Hospital on January 6, 1910, there was edema of the legs extending half-way to the knees; some cyanosis and marked dyspnea. Heart markedly hypertrophied; second aortic sound accentuated; no murmur. At that time there was no fluid in the chest. Liver much enlarged; slight ascites. A faint trace of albumin, but no casts, was found in the urine. Blood pressure: systolic, 214 mm. Hg. During the stay of twelve weeks the patient showed much improvement, which continued until July 1911, when, following undue

exertion, he had a recurrence of the foregoing symptoms. Again admitted on October 16, 1911, with signs of cardiac dilatation and right-sided hydrothorax. Following removal of this fluid the patient experienced much relief. Since then he has been aspirated at intervals of two months, during which time about 20 ounces accumulate.

Case V.—B. H. W., male, aged sixty-six years; occupation, manufacturer. Habits good, except for the immoderate use of alcoholic beverages.

On November 30, 1908, the patient came to me complaining of vertigo, dyspnea on exertion, and dimness of vision. Physical examination revealed cardiac hypertrophy and marked arteriosclerosis. Systolic blood pressure, 225 mm. Hg. Ophthalmic examination showed iritis and a retinal hemorrhage near the macula. Urinalysis: albumin, small ring; casts, several narrow hyaline and finely granular. His condition remained about the same until September 5, 1909, when he was taken acutely ill with symptoms of cardiac failure and renal insufficiency. Ten days later free fluid was discovered in the right chest and a moderate amount of transuded serum was removed. This procedure resulted in much relief as to dyspnea, but the patient gradually sank into uremic coma and died three days later.

Case VI.—V. A. C., male, aged sixty-one years; occupation, railroad inspector.

In December, 1906, I treated the patient in the Medico-Chirurgical Hospital for lobar pneumonia, which was followed by circumscribed, right-sided empyema. After the removal of the exudate, the patient recovered and remained well until January, 1910, when he began to complain of marked dyspnea, unproductive cough, and prostration. Upon his readmission to the hospital (March 5, 1910) I found the heart much dilated, blood pressure moderately elevated, Cheyne-Stokes breathing, cyanosis without dropsy, and signs of free fluid in right pleural sac. Urinalysis revealed a trace of albumin and a small number of narrow hyaline and finely granular casts. On March 15, 48 ounces of transudate were removed from the right chest; March 19, 46 ounces; March 24, 32 ounces were withdrawn. The patient experienced much relief following each operation, but on March 27, he developed facial erysipelas, and died four days later.

Case VII.—P. R., male, aged forty-two years; occupation, tailor. Moderate use of wines and tobacco; had gonorrhea fifteen years ago.

The present illness began about July 1, 1910, with pain and swelling in right leg, which, however, did not incapacitate him for work until August 15. At this time he began to complain of difficulty in breathing, at times amounting to orthopnea, associated with cough and frothy expectoration. When admitted to the

Medico-Chirurgical Hospital on October 6, 1910, dyspnea was marked, moderate arteriosclerosis present, and the right lower extremity swollen and tender, otherwise no edema. Physical examination showed signs of myocardial changes (dilatation) and right-sided hydrothorax. The transudate (18 ounces in amount) was withdrawn, but in spite of rest and vigorous cardiac stimulation the patient died suddenly three days later.

Case VIII.—P. L., male, aged sixty-five years. Besides the usual infections of childhood the patient had acute articular rheumatism when aged twenty-eight years.

The present illness began about October 19, 1910, when the patient first noticed dyspnea and palpitation on exertion. On March 16, 1911, these symptoms became more urgent. At this time the heart was dilated, second aortic sound slightly accentuated; no murmur; pulse irregular; lower extremities edematous. Urinalysis revealed a small ring of albumin and a moderate number of narrow hyaline and finely granular casts. Following rest in bed and cardiac stimulation the patient improved somewhat for a time, but early in June, 1911, the symptoms again became aggravated and a moderate right-sided hydrothorax was discovered. On June 15, 16 ounces of clear fluid, having a specific gravity of 1010, were removed. Marked temporary relief followed this operation, but the fluid rapidly reaccumulated, and aspiration was again performed on June 22. Salt-free diet was now ordered and rapid diminution in the general edema followed, but the transudate in the chest reaccumulated, though more slowly. On July 3, 23 ounces were removed from the right chest, and from this time until the date of his death, August 1, 1911, the chest remained free from fluid.

Case IX.—H. W., male, aged fifty-two years, admitted to the Medico-Chirurgical Hospital on June 15, 1911. Habits: rather excessive use of chewing tobacco and alcohol since early manhood.

The present illness began suddenly six weeks ago, when he awakened one morning in a profuse perspiration and had to gasp for breath. Under treatment by his physician, Dr. J. D. Niles, he improved and felt comparatively well until one week since, when he was again seized with marked dyspnea and vertigo, which persisted. Four days ago he first noticed swelling of feet and legs. On admission I found the patient dyspneic, cyanosed, the heart moderately dilated, physical signs of right-sided pleural effusion, and a small amount of free fluid in the abdomen. Urine negative, except for low specific gravity (1010); systolic blood pressure, 155 mm. Hg. The right chest was aspirated immediately and 10 ounces of transudate were removed. With rest in bed and the use of cardiac stimulants the patient continued to improve and left the hospital twelve days later apparently well. The patient so continued until late in July, when he had a recurrence,

and returned to the hospital, where it was found that the transudate had reaccumulated. The patient left the hospital ten days later in good condition, and there has been no recurrence of hydrothorax.

CASE X.—H. C., male, aged fifty-three years; occupation, liveryman.

Family History. Father and brother died of dropsy.

Previous History. Negative.

The present illness began June 14, 1911, when, following unusual exercise, he noticed weakness and dyspnea. He entered the Medico-Chirurgical Hospital on July 3, at which time a physical examination revealed a dilated heart, an apical murmur, with a small area of transmission, marked arrhythmia, many cardiac impulses, failing to reach the radial arteries. Liver somewhat enlarged. The urine showed a small ring of albumin and numerous hyaline casts; specific gravity, 1025. Systolic blood pressure, 106 mm. Hg.

On July 11 there was some edema of ankles. Two days later signs of free fluid appeared in the right chest. This was removed a few days later, 25 ounces being obtained. The dyspnea was at once largely relieved. Following this there was gradual improvement in his condition, with disappearance of the murmur, and the fluid showed no tendency to reaccumulate. However, on July 29, the patient sat up in bed and suddenly fell over dead.

CASE XI.—M. D. J., male, aged fifty-six years.

Family History. Rheumatism on maternal side. Had severe attack of influenza and rheumatism thirteen years ago; subject to digestive disturbances for many years.

In February, 1910, the patient began to have attacks of angina pectoris. At that time the heart was hypertrophied, arteries markedly sclerotic; systolic blood pressure, 185 mm. Hg. Under treatment the angina attacks became less frequent and gradually ceased, and the patient enjoyed fairly good health until May 29, 1911, when he began to complain of dyspnea and cough.

Physical examination on June 22, 1911, showed cardiac dilatation, arteriosclerosis, a moderate right-sided hydrothorax, and edema of the legs. Urinalysis revealed specific gravity, 1010; albumin, a small ring and casts, a moderate number of narrow and medium hyaline, finely and coarsely granular. As a result of prolonged rest in bed, salt-poor diet, purgation, and cardiac stimulation the fluid gradually disappeared, with marked improvement in his general condition. Since July, 1911, there has been no return of the hydrothorax, and the patient has remained free from symptoms.

CASE XII.—B. E., male, aged fifty-one years. Mother deceased of Bright's disease, and father of heart disease. Nine years ago had ureteral calculus (left-sided) removed. Moderate use of alcohol; tobacco to excess.

The present illness began in October, 1910, with dyspnea, dry

cough, but no edema; as result of treatment he improved and felt well until three weeks ago, when, following undue exertion, the above symptoms recurred in an aggravated form. On November 7, 1911, when I first saw him, there was much distress of breathing, heart dilated and hypertrophied, some edema at bases of lungs and signs of right-sided pleural effusion without general dropsy; systolic pressure, 172 mm. Hg.; arteries palpable. Small albuminuria and the presence of narrow hyaline and granular casts. Patient admitted to Medico-Chirurgical Hospital, where he remained five months at complete rest, with the use of a salt-poor diet, saline laxatives, and cardiac stimulants. Heart stimulants, however, aggravated the dyspnea until transudate had been removed. Later developed edema of legs. After six tappings the fluid failed to recur and external edema disappeared. Patient left the hospital on April 6, 1912, in fairly good general condition. On July 1 received statement from his home physician reporting a sudden recurrence of cardiac dilatation after too much exertion, proving rapidly fatal.

Case XIII.—T. F. L., male, aged seventy-four years. The patient had acute articular rheumatism when aged thirty-nine years.

The present illness began two years ago, when the patient first noticed arrhythmia associated with dyspnea on exertion. When first seen by me on December 24, 1911, there was orthopnea, legs slightly edematous, vessels decidely sclerotic, heart action irregular, heart dilated, a faint apical systolic murmur, and marked right-sided hydrothorax, which had been overlooked by the two attending physicians. Urinalysis showed a faint trace of albumin and occasional hyaline casts. Systolic blood pressure, 118 mm. Hg.

After the last of six tappings within three months the fluid showed no tendency to recur and the murmur had disappeared. During this period the patient was given a non-nitrogenous, salt-poor diet, purgatives, and cardiac stimulants. The patient gradually improved and felt well until the last of June, 1912, when he had an apoplectic stroke, which proved fatal.

Case XIV.—S. J., male, single, aged twenty-one years; weight, 135 pounds; height, 5 feet 8 inches; occupation, laborer.

Family History. Negative. Five years ago was squeezed between two railroad cars, sustaining some internal abdominal injuries. Habits good.

Two years after the above-mentioned trauma was received, dyspnea and abdominal distention appeared. February, 1912, the abdomen was tapped and 20 quarts (patient's statement) of ascitic fluid removed. During the next three months paracentesis abdominis was performed three times.

When admitted to the Medico-Chirurgical Hospital on June 22, 1912, there was urgent dyspnea; cyanosis and arrhythmia; large amount of free fluid in right chest; heart dilated; no murmur

audible; excessive ascites; liver enlarged; very slight edema of lower extremities. The urine was normal excepting the presence of a few hyaline casts. Blood pressure: systolic, 115; diastolic, 100 mm. Hg. On June 24, 10 quarts of transudate were removed from the abdomen. The right chest was frequently aspirated and also the abdomen. Following each operation the patient was much relieved, but both the pleural and ascitic fluids showed a tendency to reaccumulate rapidly. Shortly before leaving the institution a small amount of fluid was discovered in the left pleural sac; this however, was insufficient to necessitate aspiration. The patient left the hospital on August 7, 1912, and died three months later.

Case XV.—J. L. J., male, married, aged sixty-four years; weight, 144 pounds; height, 5 feet 8 inches; occupation, merchant.

Family History. Negative. About eitht years ago had some pulmonary trouble; since then has been spending winters in Florida. Habits good, although he worked hard under nervous tension.

The present illness first manifested itself about two years ago, when slight dyspnea on exertion was noticed. Early in July, 1912, dyspnea became constant, and at times amounted to orthopnea. When I first saw the patient on July 12, 1912, his breathing was very labored and signs of moderate arteriosclerosis and cardiac dilatation were present. At the apex was heard a faint systolic whiff. Signs of bilateral hydrothorax, which had been overlooked, of considerable extent were elicited. At this time slight edema of the ankles was noted; later this became marked and extended to the hips. Urinalysis revealed a small ring of albumin, a moderate number of narrow hyaline and finely granular casts, specific gravity of 1022, and twenty-four-hour quantity, 20 ounces. Systolic blood pressure, 162; diastolic, 140 mm. Hg.

The next day the patient was aspirated and 2 quarts of fluid were removed from each pleural sac. Reaccumulation however occurred, and the operation was repeated on August 3, 13, and 21, with a similar result. Following the last operation, absolute rest in bed was enjoined (owing to extreme nervousness and dyspnea this measure had not previously been practicable), and a salt-poor diet, cardiac stimulants, and saline laxatives were ordered. Under this treatment there was marked improvement; the edema of the legs rapidly disappeared and the chest remained practically free from fluid, and cardiac compensation was reëstablished. After a rest in bed of two weeks the patient was permitted a limited amount of exercise; his favorable condition persisted until early in November, when as a result of unwonted physical exertion there was a recurrence of the previous symptoms, with evidences of uremic intoxication superadded. Following the withdrawal of the pleural transudate and the institution of other therapeutic measures,

improvement gradually ensued. The patient's present condition is quite encouraging, although aspiration of the chest becomes necessary at intervals of about three weeks.

CASE XVI.—W. G., male, aged fifty-nine years; weight, 190 pounds; height, 5 feet 8 inches; occupation, shipper in brewery. Father deceased of heart disease; mother of kidney disease. Intemperate use of coffee, tobacco, and alcohol.

Began about ten years ago, with dyspnea, at intervals palpitation and headaches. On admission to hospital on August 13, 1912, he showed a tendency to stupor, was dyspneic and cyanosed, heart dilated with relative mitral systolic murmur, second aortic sound accentuated. Signs of free fluid in right pleural sac. The systolic blood pressure, 185 mm. Hg. The urine contained a medium ring of albumin and a moderate number of hyaline casts; specific gravity, 1008. Examination of the eye-grounds by Dr. J. A. Brophy revealed albuminuric retinitis and recent hemorrhages in both eyes. Removal of the fluid was followed by relief, but patient gradually became comatose, dying four days later. No autopsy permitted.

I am much indebted to Dr. H. Leon Jameson for valuable aid in connection with the clinical records detailed above.

CONGENITAL ATRESIA OF THE DUODENUM.

BY ARCHIBALD L. MCDONALD, A.B., M.D.,
ATTENDING PHYSICIAN TO CHILDREN'S HOME, DULUTH, MINNESOTA.

INSTANCES of congenital atresia, or complete obstruction of the gastro-intestinal tract, are sufficiently infrequent to warrant the detailed report of individual cases when confirmed by operation or autopsy. The number of recorded cases is by no means large, though no doubt, some are not reported and many pass unrecognized in the absence of postmortem examination. Because of this assumed rarity the condition is not appreciated and the clinical diagnosis is not considered as probable. These malformations are of interest: (1) as a clinical entity, difficult of recognition, and, as to prognosis, practically hopeless; (2) in its clinical and etiologic relationship to spasm and conditions of partial obstruction of the tract; (3) the possible embryologic factors causing such abnormalities are of theoretic interest.

The following case which occurred in my practice is a good example of a complete atresia, with loss of continuity of the duodenum. Baby O.; mother, aged twenty-four years, always well; no miscarriages; two children, aged five and three years respec-

tively. Pregnancy normal; labor apparently precipitated by overwork about two weeks before expected date. Membranes ruptured, with escape of considerable fluid, at 7 A.M., December 11; head engaged in L. O. I. A. by 11 A.M.; child born at 9 P.M., after about one hour of severe pains. Placenta and membranes normal. Though membranes were said to have ruptured in the morning and much fluid escaped during the day it was found in the second stage of labor that a membrane and bag of water preceded the head. This was ruptured, followed by a gush of amniotic fluid. Though not striking at the time, there was evidently an hydramnios, judged by the excessive amount of fluid. This is said[1] to frequently accompany such malformations in the fetus. However, hydramnios is frequently observed, and its occurrence alone would hardly lead one to expect a gastro-intestinal abnormality. Child weighed six pounds eight ounces, and was apparently normal in every respect.

December 12. Child had vomited in the night, had nursed well, and was hungry. Passed some urine, but no bowel movement; continued to vomit at irregular intervals during the day.

December 13. Patient passed slightly colored meconium; nursed well, but continued to vomit; was restless and slept poorly; no urine observed.

December 14. Baby appears emaciated and slightly jaundiced; abdomen distended; vomited large amounts of fluid, containing no bile and in no relation to feeding. Soapsuds enema brought away slightly colored stool. Nursing discontinued and albumin water substituted.

December 15. Child still vomiting; is restless and sleeps poorly; no stools or urine; enema comes away clear. Stomach tube drew off nearly a pint of creamy fluid not bile stained. One could not be sure that all contents were removed, but the washings came away clear and the abdomen was much less distended than before. A diagnosis of pyloric stenosis, with probable complete obstruction, was made, and a hopeless prognosis given. Child rested better for some hours, but became restless during the night and died at 7 A.M.

December 16. A partial autopsy was permitted and only the abdominal condition will be described, since as near as could be determined other parts were normal. On opening the abdomen the distended stomach and duodenum were most prominent, but in normal position, as were the other viscera and omenta. The stomach was dilated to three times its normal size, and the duodenum was much larger than the stomach. There was a definite constriction at the pylorus, which was, however, patent. The duodenum was greatly dilated, but could be traced through its

[1] Little and Helmholz, Johns Hopkins Hosp. Bull., July, 1905.

normal course to a blind sac behind the stomach and transverse colon, where it ended, there being no communication or connection with the lower intestine. Rectum and colon were small and

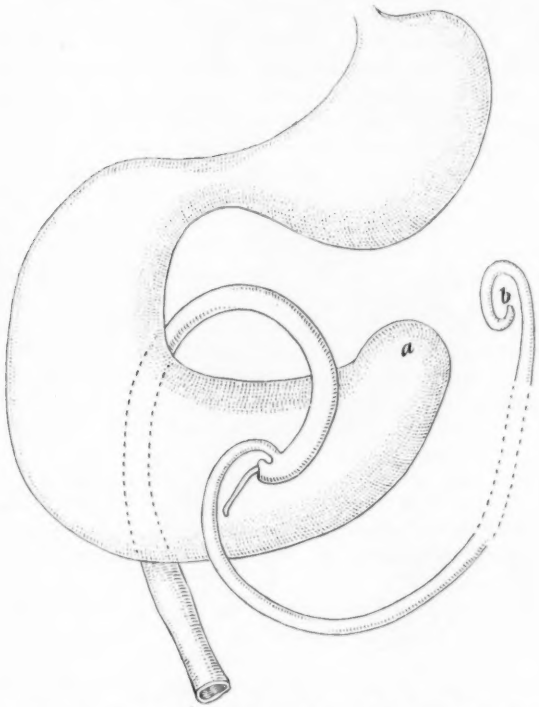


FIG. 1.—*a*, blind end of duodenum; *b*, upper end of jejunum buried in fold of mesentery.

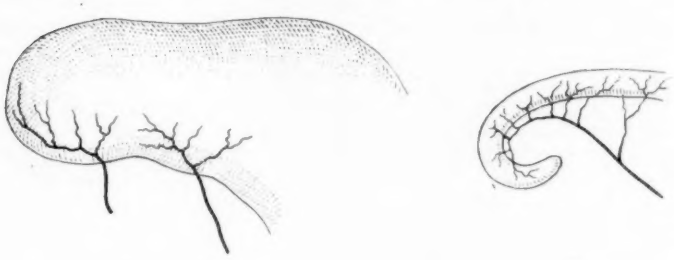


FIG. 2.—*A*, lower end of duodenum with blood supply from superior pancreaticoduodenal artery. *B*, upper end of jejunum buried in mesentery. Blood supply from inferior pancreaticoduodenal artery.

collapsed, the latter extending across the upper abdomen, with the cecum in about its normal position. Appendix long, with normal mesentery. Small intestine contracted and cord-like, being barely permeable. Its mesentery was normal, and no definite constriction was found until reaching the upper end of the jejunum in the region of Treitz's fossa, where it ended in a blind sac covered by a fold of peritoneum. Liver normal in size and position. Gall-bladder distended with bile. Ducts could not be traced to the duodenum. It is probable that no communication existed, as no bile was present in the vomitus or contents of the distended stomach or duodenum, and jaundice was present. We had then a complete atresia, with obliteration of a portion of the tract in the region of the duodenojejunal junction.

Such malformations are not extensively discussed in text-books or even in the literature, except when mentioned as unusual congenital anomalies. There are, however, a few extensive articles giving descriptions of such cases and reviewing the literature, to which I shall refer.[2]

Defects may be found at any point in the gastro-intestinal tract, but are more common at the following sites and in the same order of frequency: (1) pylorus and duodenum; (2) rectum and anus; (3) ileocecal region; (4) at the attachment of Meckel's diverticulum; (5) the flexures of the large intestine. Atresia and stenosis of the rectum and anus really belong in a different group and will not be considered. Little and Helmholz were able to collect only 27 cases of atresia of the duodenum above the papilla of Vater, including their own. Spriggs adds one of the duodenum, and reports several of the tract lower down. Clogg refers to the following collections of cases: Silberman, with 24 cases of atresia of the duodenum, and 30 of atresia, and 3 of stenosis of the jejunoileum. Schlegel and Braune, with 89 cases of atresia and stenosis of the entire tract, Cordes,[3] with 48 cases of atresia and 9 of stenosis of the duodenum. These references are also given by Little and Helmholz, so it is fair to assume they include most of the cases in the literature. Several areas of atresia have been reported in a single individual.

Unless the case is studied with the possibility of such a condition in mind the diagnosis is not made until treatment is hopeless and most often is missed entirely. Many such cases would be classed as malnutrition or spastic stenosis, unless an autopsy confirmed the diagnosis of atresia. Certain symptoms and signs are suggestive, and if carefully studied will enable one to make a corect diagnosis, possibly early enough to warrant surgical intervention. (1) Persistent and characteristic vomiting; (2) constipation and

[2] Little and Helmholz, Johns Hopkins Hosp. Bull., July, 1905; H. S. Clogg, Lancet, December 24, 1904; N. J. Spriggs, Lancet, January 8, 1910.

[3] Archiv f. Pediatrics, 1901.

character of the stools; (3) distention of abdomen, visible peristalsis; (4) anuria, emaciation, jaundice, and other malformations; (5) hydramnios in mother. Vomiting is a constant and important symptom, and may include amniotic fluid, and everything taken by the mouth. It has no necessary relation to feedings, the frequency and amount depending upon the size of the cavity above the stenosis. Bile will be present or absent as the obstruction is above or below the papilla of Vater. Most characteristic is the vomiting of drugs or food given a day or so previously. A stomach tube should be used early in suspected cases, and will demonstrate the size of the viscus. Such findings are often the first indication of an organic stenosis. Vomiting in the newborn is so common that it is often not properly appreciated, but in these cases the duration is short, five or ten days or less, and the progress is rapidly fatal, so that the symptoms should be carefully studied from the beginning. If the stomach is greatly dilated the abdomen will be prominently distended and splashing sounds may be elicited. There may be one or more spontaneous passages of meconium, or an enema may bring away some intestinal contents. In such cases, as with mine, one feels that the condition of the bowels is temporary and that the next stool will be normal. However, such stools rarely contain bacteria, and never, if the obstruction is complete, drugs or digested food. Bile pigments are absent if the obstruction is below the papilla. There may be complete obstipation, which would indicate an obstruction lower in the large intestine. Distention of the abdomen, with visible peristalsis, will be suggestive when present.

Anuria is usually a marked symptom, due to the fact that no fluid is absorbed from the stomach. Emaciation is rapid and progressive, as the child is receiving no nourishment. Daily weighings will show a rapid and regular loss of weight. One might expect to find some form of tetany in these cases, due to the absorption of toxins from the stomach and duodenum; but it was not present in my case, neither is it mentioned in the literature. Jaundice is mentioned in some instances, and was marked in my case. Other congenital deformities might suggest organic obstruction of the intestinal tract in the presence of symptoms. Hydramnios in the mother is said to frequently accompany, but as already stated is too common to alone suggest deformity in an otherwise normal child. Most of the symptoms are exaggerations of those of malnutrition, and often do not attract great attention during the first day or so. The picture rapidly becomes striking, and if one has followed it carefully a diagnosis of pyloric or duodenal obstruction should be made.

More important and also more difficult is the differential diagnosis between (1) spasm of the pylorus; (2) congenital hypertrophic stenosis, and (3) congenital atresia, with complete obstruction.

Spasm of the pylorus may be present shortly after birth, but often does not cause symptoms for some days, weeks, or even months. Usually a certain amount of nourishment passes into the intestine, so the emaciation is not as rapid as is the case in absolute obstruction. The stools show the presence of bacteria and digested food. Hypertrophic pyloric stenosis may give marked symptoms during the first few days, but this also may be delayed for some weeks, and the course is not as rapidly downward. Unless the stenosis forms an absolute obstruction the stools will contain bacteria and digested foods. A palpable tumor would, of course, suggest hypertrophic stenosis. Vomiting is a marked symptom in both the above conditions, but emaciation and malnutrition are slower in their development. In a case of congenital atresia, or complete obstruction, the symptoms must be more marked and rapidly progressive than in those of relative stenosis from spasm or hypertrophy.

The prognosis is bad in all cases of even relative obstruction in the newborn because of the difficulty of nourishing the child, and is practically hopeless in case of complete congenital atresia. Hypertrophic pyloric stenosis is well recognized and discussed in the literature. There are several reports of successful surgical treatment of these cases and a few instances of medical treatment, though in the latter group the diagnosis must remain in doubt. In the complete obstruction or atresia the condition is absolutely hopeless unless the continuity of the intestinal tract can be reestablished by surgical procedure. So far as I can find in the literature no such successful operation has been reported, though many have been attempted. There are several factors which are unfavorable to a successful outcome. The diagnosis is rarely made before the child is emaciated and in poor condition to undergo operation; the intestine in the newborn is normally small, rendering an anastomosis difficult at best; the portion of intestine below a congenital stenosis or atresia has been empty and collapsed for months, and perhaps has never been patent. Other obstructions may be present lower in the tract. In my case the distal portion was almost a solid tube, with only a small lumen. Nevertheless in view of the absolutely hopeless prognosis if let alone, operative treatment would be indicated as early as the diagnosis of complete obstruction is made. It is to be hoped that early diagnosis and improved operative technique may save a baby otherwise doomed to starvation. The practically fatal prognosis should be explained to the parents before a surgeon is asked to take the responsibility of operation. Little time should be spent in searching for the obstruction, as the condition of the tract above and below will be sufficient to indicate the sight. Gastro-enterostomy is the operation of choice. If the obstruction is much lower an enterostomy may give temporary relief, and the infant brought into better condition to undergo a final operation.

It is stated that most congenital atresias develop before the fourth month of intra-uterine life, since bile is not found in the distal portion of the intestine. This would fix the time of development before certain important embryologic changes have taken place and to some extent would limit the possible causal factors. Might it not be possible, however, for a certain amount of bile to be absorbed from the fetal intestine into the portal circulation, leaving little trace of its presence. Some of these obstructions might therefore be supposed to occur at a later period. The theories advanced to explain these anomalies are numerous and interesting. The inflammatory theory explains them as being due to adhesions following fetal peritonitis. Ascites has been described in some cases of congenital obstruction, but extensive adhesions are rarely found. Fetal peritonitis, especially in the early months, is not a common finding, and must be limited to the tuberculous or syphilitic forms. It is improbable that a fetus affected with syphilitic peritonitis of sufficient intensity to cause atresia of the intestinal tract would go on to full term, much less develop as an otherwise healthy child. Few if any of these cases have shown signs of congenital syphilis in life or at autopsy. Fetal tuberculous peritonitis is rarely found, and it is hard to believe that only a part of the tract could be involved, leaving no other evidence of the disease. Fetal inflammation may explain isolated cases, but will not cover the majority of them. Pearce-Gould, in 1882, described a case in which the cecum and ascending colon were filled with a plug of cheesy mucus adherent to the intestinal wall. This instance could be explained on the intussusception theory, as advanced by Chiairi, who found an atresia 15 cm. above the cecum, with a cylindrical intussusception in the distal portion of the colon. This might explain cases of atresia of the lower bowel more often than is evident at first thought as the necrotic bowel may be disintegrated or absorbed, leaving little proof of the original condition. However, it can hardly explain such anomalies of the pylorus or duodenum. Fetal volvulus, adhesions of Meckel's diverticulum, and an inclusion of a part of the intestine in the closing umbilical ring may cause kinking, occlusion, or atresia of the small or large intestine, but probably do not affect the duodenum. Bland Sutton, in 1889, advanced the idea that most of these malformations were due to errors in development, the most frequent locations of which correspond with the sites of important embryologic events. For example, the most frequent locations of atresia are: (1) pylorus and duodenum, the site of a complicated rotation as well as the outgrowth for the liver and pancreas; (2) the ileum, especially at the attachment of Meckel's diverticulum; (3) ileocecal region, where there occurs a rotation and what is essentially an outgrowth of the sacculated cecum and appendix; (4) the region of the flexures is a less frequent site of malformation and of less importance embryologically. The most

important embryologic changes in the intestinal tract occur in the region of the pylorus and duodenum, but normally result in a patent continuous tube. The duodenum is relatively fixed at the pylorus and at its junction with the jejunum, and the tract undergoes a double rotation about these points. The outgrowths for the liver and pancreas make in all a complicated picture, which, however, normally results in a patent continuous intestinal canal.

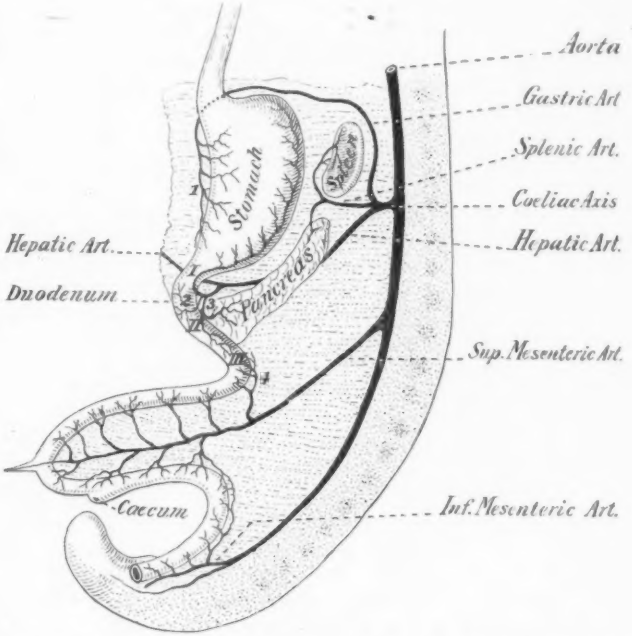


FIG. 3.—Arteries of alimentary tract in human embryo at six weeks: 1, gastric artery; 2, pyloric artery; 3, superior pancreaticoduodenal branch of hepatic; 4, superior pancreaticoduodenal branch of superior mesenteric. *I*, anastomosis between gastric and pyloric branches of hepatic; *II*, anastomosis between pyloric and superior pancreaticoduodenals; *III*, anastomosis between superior pancreaticoduodenal branch of hepatic and inferior pancreaticoduodenal branch of superior mesenteric. (Modified from Kollmanns.)

Likewise at the other sites mentioned the development offers opportunity for deviation from normal and possible occlusion of the canal. However, obstruction does occur at regions where no very complicated event takes place, and must be explained on other grounds. Also some additional factor is required to explain the deviations from normal in the above-mentioned sites. This has been suggested by Joubalay in the idea of congenital vascular anomalies. Little and Helmholz quote a case of atresia of the

duodenum above the papilla in which there was an absence of the superior pancreaticoduodenal artery, and in their own the superior pancreaticoduodenal, pyloric, and left gastroepiploic arteries were wanting. The blood supply of the fetal gastrointestinal tract below the diaphragm is from three main aortic branches which anastomose freely. (See Fig. 3.)

That of the pylorus and duodenum is from three sources whose branches anastomose freely: (1) a branch from the gastric, which anastomoses with a branch from (2) the hepatic, the pyloric. The hepatic also gives off the gastroduodenal, which divides into the right gastroepiploic and superior pancreaticoduodenal. This latter anastomoses freely with the inferior pancreaticoduodenal from the (3) superior mesenteric. We have then a blood supply from three sources which anastomose freely, and should normally secure efficient nutrition to all parts. Vascular anomalies are not infrequent, and we have here several places where an occlusion or absence of a normal branch might interfere seriously with development of the part, thus leading to partial or complete atrophy. This would be marked by kinking, stenosis, or obliteration of a part, with complete atresia. This is most likely to occur at the pylorus, where branches of the gastric and hepatic anastomose, and at the lower portion of the duodenum supplied by branches of the superior and inferior pancreaticoduodenal. Such vascular anomalies are perhaps most frequent in the duodenal region, but the same possibility exists at many points in the tract. The branches of the vasa intestina tenuis of the superior mesenteric are essentially terminal, and there is little anastomosis between them. Occlusion of one or more of these offers an explanation of atrophy of the small intestine. Whether the fusion of the fetal "intestinal segments" as described by Mall present sites of possible stenosis or occlusion is also an interesting question. A study of the vascular supply to the ileocecal region and its development offer several points where the chain may be broken and an area left without sufficient blood supply.

Such factors then as inflammatory adhesions, volvulus, occlusion by intestinal contents, may explain an isolated case, and this has been apparently demonstrated by careful autopsy dissection. However, there remains a large number not satisfactorily explained by any of these causes. The fact that the majority of these malformations occur at regions where important embryologic events take place renders their explanation as due to developmental error plausible. Together with this we must consider the relation of vascular anomalies as exceedingly important. In fact, I believe these two factors are interdependent, and will explain most of the cases under discussion.

RECOVERY FROM TUBERCULAR MENINGITIS, WITH REPORT OF CASES.

By Robert L. Pitfield, M.D.,

PHYSICIAN TO ST. TIMOTHY'S AND GERMANTOWN HOSPITALS, PHILADELPHIA.

McCarthy in the *Phipps Institute Reports* for 1908 remarks "that evidences of recovery in tuberculosis of the nervous system is as rare as it is common in the lungs." That there is a retrograde process in tuberculosis of the glands, bones, peritoneum, and lungs is, of course, a trite observation.

In the nervous system occasionally tuberculous processes calcify, and meningeal infections become fibrous, quiescent, and finally heal. That such is the case in meningitis of tubercular origin has been a matter of comment by quite a number of writers, notably by Martin, in *Brain* for 1908, who has collected a series of cases in which recovery took place. In all text-books the prognosis is put down as grave or always grave. Dieulafoy mentions but one case that recovered, but he evidently had not scanned the literature thoroughly, because in his own country at the time at which his book was written there were records of at least six or seven cases of undoubted tubercular meningitis followed by recovery. But even the most optimistic must agree that it is always best to tell the family of one stricken with this disease that the outlook is practically hopeless; nevertheless, one must do so guardedly, and follow out certain lines of rational treatment, to be detailed later, in the hope that the lesions may be limited to the cortical membranes of the brain, without exudation of fibrin or purulent matter, and may not extend into the ventricles.

There are, of course, several meningeal diseases closely simulating tuberculosis, and these must be considered in several ways. These are, notably: (1) Quincke's disease, in which there is simply an excess of cerebrospinal fluid, chiefly in the ventricles, not produced by microörganisms and not infectious; (2) influenzal meningitis, simulating it closely, (3) and the meningismus of typhoid. Hereditary syphilis in the young, too, may resemble meningitis of tubercular origin. From the fact that Quincke's disease, which is not of parasitic origin, and therefore probably not toxic in the character of the serous exudate, can simulate true meningitis, it is evident that simple pressure within the ventricles and hence to the skull may cause nervous symptoms, chiefly cephalalgia in both disorders. In meningitis the added poisons and the multiple tubercles massed in nerve trunks and fibrous purulent exudate add much to the pathology and to the gravity of the prognosis.

A typical case of influenzal meningitis is the following: A young grocer, aged twenty-seven years, married, father of one healthy

child, was one evening suddenly seized with a violent headache. This did not yield to any of the ordinary headache remedies, so that by morning morphine was required. Then it was seen from the retraction of the neck, intense pain, retracted belly, tâche cérébrale and Kernig's sign, fever, photophobia, and delirium, that meningitis was present. Accordingly, a lumbar puncture was done and 70 c.c. of clear liquid were drained off. This contained a fibrin clot, lymphatic cells exclusively, and no organisms. The headache immediately disappeared, Kernig's sign and rigidity of neck did so after ten days. He gained weight and resumed business. Two months afterward a second attack necessitated another puncture. He then had double vision on looking down and marked abduction of the eyes; jerking of the eyeball was demonstrable. Photophobia, increased knee-jerks, plantar flexion of the toes, tâche cérébrale, rigidity of the neck on the right side appeared. At this time headache did not seem so intense, and there was not so much fever. In removing 35 c.c. of fluid the double vision and fulness of the head disappeared during the operation, and the patient became markedly better. The fluid was limpid, clear, but contained some minute bacilli, probably influenzal. A guinea-pig injected with the fluid recovered. In twenty days all symptoms of any disorder promptly disappeared, save some stiffness of the neck. This was probably a case of influenzal meningitis, with lymphocytosis, cured by lumbar puncture and urotropin, which was freely given. There was no history of syphilis in the case, and the Wassermann test was negative.

The following is the history of a case of tubercular meningitis with recovery:

J. S., aged fifty years, linen draper, was admitted to St. Timothy's Hospital with every symptom of meningitis, from which he had suffered for four or five days. He had had such an intense headache that be begged to be shot, and indeed a revolver in his room had to be removed for fear that he would do so. When admitted he was delirious and had a retracted neck, scaphoid belly, and tâche cérébrale. A lumbar puncture was done under chloroform and 80 c.c. of fluid were withdrawn. He was a very tall man, measuring over six feet four inches, but was far from robust. In each apex there was evidence of tuberculosis, which from his history had existed for some time; in every way his history was that of one who had a low-grade phthisis for ten years. The present attack came on with violent vomiting and unendurable headache, also pains in his arms and legs. Kernig's, Oppenheim, and Babinski's signs were all present markedly. Pupils were contracted and rigid. On attempting to flex his neck after the lumbar puncture all the major joints flexed, and he groaned.

The lumbar puncture was easily performed, the fluid spurted out, and was quite bloody, due to the puncture of a small vein. This fluid contained three acid-fast bacilli, which in view of the presence

of tuberculosis of the lungs were assumed to be tubercle bacilli. No animal injections were made. A cytodiagnosis was not attempted because of the admixture of blood. His pulse was 70; respiration, 26 and jerky; temperature, 99°. His leukocytes numbered 6200; red cells numbered 4,200,000; hemoglobin, 75 per cent. His urine was bloody, and contained albumin and casts. Blood pressure: 128 systolic; 88 diastolic. Marked improvement followed the withdrawal of the fluid. His spastic symptoms slowly receded. His mind became clear in a few days and his headache disappeared. He was given forced diet and urotropin, 30 grains a day. After three weeks' stay in the hospital, to the amazement of all, he suddenly got up, asked for his clothes, and went home. He then had some rigidity of his neck and some evidences of Kernig's phenomenon. After two years he is able to conduct his business and has no evidences whatsoever of any meningitis. In this instance the fact that the patient was an adult and long infected with a low-grade tuberculosis no doubt contributed much to his recovery.

Perhaps the most interesting case of recovery after tubercular meningitis is that reported by Rumple (quoted by Martin), who exhibited at the Aerztlichen Verein, Hamburg, the brain of a boy who died of phthisis. At the age of nine the boy had an acute attack of meningitis, in which there was retraction of the neck and hyperesthesia, absence of knee-jerks, bilateral optic neuritis, ptosis, and palsy of the external recti. Lumbar puncture gave a fluid in which tubercle bacilli were found. The boy, who was treated with repeated lumbar punctures, recovered, and though for some months was dull and stupid, he eventually retained his normal mental condition and was able to keep pace with other children of his age at school, while all physical signs had disappeared. Four years his health continued good, he then developed a tubercular abscess in the axilla, followed by phthisis; death took place eight years after the onset of the meningitis. At autopsy it was found that the membranes over the fissure of Sylvius on either side of the brain were thickened and glued together, but miliary tubercles were no longer visible. Not only was there a confirmation during life of the presence of tubercular meningitis, by the finding of tubercle bacilli, but also post mortem, at which the site of the old infection was discovered and definitely identified. Martin sums up the following requirements to be met in making a diagnosis of tubercular meningitis followed by recovery:

1. Clinical evidence of tuberculosis elsewhere in the body and history of exposure.
2. Differential count of cells in the spinal fluid (meaning an excess of lymphocytes).
3. Presence of tubercles in the choroid.
4. Tuberculin reaction.
5. Demonstration of bacilli in the fluid by staining, inoculation, and culture.

6. Postmortem examination after recovery or long remission and death by some other agency.

If tuberculosis of the meninges be but a local manifestation of a generalized infection (miliary tuberculosis) then the likelihood of recovery is of course minimized. That it is likely under such circumstances, is evidenced by cases reported by Lunn, (1) Dujardin-Beaumetz (2), Thornalas (1), and Brooks (2), in all of these tubercles were seen in the choroid, indicating a generalized infection. Tuberculin reaction, of course, confirmed the diagnosis, but it does not of necessity prove that there may not be some other remote latent infection which is the cause of the reaction. Postmortem examination and clinical diagnosis of tuberculosis elsewhere are, of course, convincing confirmation that the disease is tuberculosis.

In the case of a young woman, recently under the care of the writer, who had severe retractions of the neck, with pain, photophobia, increased knee-jerks, tâche cérébrale, tuberculous apex, bilateral pleurisy, high fever, and definite history of exposure to tuberculosis in her home, lumbar puncture revealed nothing and did not alleviate symptoms a particle. A later development of iritis and arthritis of the elbow compelled a revision of diagnosis to rheumatism of the spine, pleura, joints, and eye, showing that mere dependence upon the previous existence of a latent tuberculous lesion for confirmation was misleading and not dependable. Cytodiagnosis is also misleading. Porat reports cases showing that it may be valueless. Forbes, who examined 80 cases, 70 being verified by autopsy or by finding tubercle bacilli, found 51 had an excess of lymphocytes, 5 had an excess of polymorphonuclears; in 4 the proportion was equal. In tuberculosis of the brain not communicating with the meninges the fluid was normal. Goggia found an excess of lymphocytes in a meningococcus infection. Mutzner concludes, from a study of a series of cases with postmortems, that a lymphocytosis is not by any means diagnosed, and reports cases with an excess of the many nuclear forms of leukocytes.

In 797 cases collected by Martin for the London Hospital of unconfirmed tubercular meningitis, 16 (or 2 per cent.) recovered. In Vienna 1369 cases were reported in which recovery took place in 6 (or 4.4 per cent.). Thus in a total of 2166 cases, 22 (or about 1 per cent.) recovered. These, of course, were diagnosticated from mere clinical symptoms. Barlow, also quoted by Martin, states that if the tuberculous process be limited to a part of the surface of the brain there is a possibility of recovery, but if the disease, becomes generalized so that besides the invasion of the pia mater there is an extensive meningo-encephalitis, with or without hydrocephalus, the chances of recovery are practically nil.

McCarthy in several *Phipps Institute Reports* found frequent evidences of healed tuberculosis of the nervous system in subjects that had finally died of phthisis.

TRUE CASES.

Case.	Autopsy.	Bacilli found.	Tuberculosis elsewhere in body.	Cytodiagnosis.	Tubercles in choroid.	Clinical diagnosis.	Tuberculin.	Tuberculous history.	
Treyhan.	None, recovered	Yes	..	..	None	Yes	None	..	Choked disk.
Henkel, 17 yrs.	None, recovered	Yes	..	..	..	Yes	..		
Gross, 20 yrs.	None, recovered	Yes	Yes	Excess polys.	..	Yes	..	..	Developed phthisis 8 months after.
Stařk, 44 yrs.	None	Yes	..	..	None	Yes	..	..	Optic neuritis.
Barth, 3 yrs.	None	Yes	None	None	None	..	..	..	Recovered in 6 months.
Stiles.	None	Yes	None	..	None	Yes	..	..	Recovered after decompression.
Stiles.	None	Yes	None	..	None	Yes	..	..	Recovered after decompression.
Alanzino.	None	Yes G. P.	None	Yes	..	Yes	..	..	Recovered in good health, 6 months.
Tedeschi, 14 yrs.	None	Yes	None	Yes	..	Yes			
Jemma, 3½ yrs.	None	Yes	None	Yes	..	Yes	..	..	After 3 years, good health.
Garciso, 8 yrs.	None	Yes	None	Yes	None	Yes	..	..	Well 12 months after.
Claisse, 30 yrs. Abramc.	None	Yes	None	Yes	..	..	..	..	Satisfactory recovery.
Dufour.	Yes	..	..	..	..	Yes	..	..	Old tubercles found.
Dufour.	Yes	..	..	..	..	Yes	..	..	Old tubercles found.
Vaquez-Digne.	..	Yes G. P.	Yes	Yes	..	Yes			
Rossini.	..	Yes G. P.	..	..	..	Yes	..	..	Complete recovery.
Janssen, 19 yrs.	Yes	..	..	..	..	Yes	..	..	Meningitis 3 years before death from phthisis.
Politzer.	Yes	..	..	..	..	Yes	..	..	Old lesions and new lesions, 3-year interval between attacks.
Schwalbe.	Yes	..	..	..	..	Yes	..	..	Both died of diphtheria.
Schwalbe.	Yes	..	..	..	..	Yes	..	..	Evidence of old tubercles found at autopsy.
Carrington.	Yes	..	..	..	..	Yes	..	..	Died of psoas abscess.
Leube, 24 yrs.	Yes	..	..	..	..	Yes	..	..	Died of phthisis and recurrent meningitis. Old tubercular lesion of brain found from previous attack.
Riebold, 6 yrs.	None	G. P. Yes	..	..	..	Yes	..	..	574 c.c. drawn in punctures.
Rumpel, 17 yrs.	Yes	G. P. Yes	Yes	..	..	Yes	..	..	Died of phthisis 4 years after recovery from meningitis.
Lunn.	None	..	..	..	Yes	Yes	..	..	Meningitis followed by recovery.
Dujardin-Beaumetz.	None	None	None	..	Yes	Yes	..	..	Recovery; diagnosis based on choroid tubercle.
Thornallas.	None	None	Yes	None	Yes	Yes	..	..	Tubercles seen in choroid; disappeared on recov'y.
W. T. Brooks, 4 yrs.	Yes	Yes	Yes	Yes	Yes	Yes	Yes	..	Autopsy showed old healed meningitis; subsequently died after a long period.
Pitfield, 55 yrs.	None	Yes	Yes	Yes	None	Yes	None	Yes	Recovery, 3 weeks; old tuberculous lesion of the lungs.

I am able to append 29 cases of undoubted tuberculosis, mostly collected by Martin, in which recovery followed and 8 others more doubtful. In 10 autopsy confirmed the early diagnosis. In 18

tubercle bacilli were detected and in 5 of these guinea-pig inoculation confirmed the diagnosis. In 4 cases, tubercles of the choroid were seen. In 7 demonstrable tuberculosis elsewhere was noted.

In view of the fact that recovery can take place in perhaps 1 in 200 cases, steps should be undertaken actively to facilitate such a possibility. The patient should be put in a quiet, airy, dark place and kept as free from annoyance as possible. Lumbar puncture should be performed at once. The writer had a case, a boy, aged five years, from whose spine 40 c.c. of fluid were removed every other day for two weeks, with marked amelioration in the symptoms. Forced feeding through nasal tube, with milk and eggs, should be done. Morphine administered for pain and as a sedative, if respiration is not embarrassed thereby, and because free formaldehyde is found in the spinal fluid after the ingestion of urotropin the latter drug should be given freely. I have found, after giving it for one day, that formaldehyde can be detected in the fluid with iron and sulphuric acid; and because Raw has reported recovery in two cases in which tuberculin was used, I would advocate one or two injections of this remedy.

One case was most skilfully operated on by Dr. George Müller, who decompressed and punctured the left lateral ventricle, withdrawing about four times the normal amount of fluid, the child ultimately dying of respiratory palsy, having lived for three weeks after the diagnosis had been made and gaining five pounds by forced feeding. Why decompression is not fraught with better results in this disease is probably due to the involvement of the ventricles. The unrelieved distention is due to the choking of the canal leading from the third to the fourth ventricle.

AN INTENSIVE STUDY OF THE EPIDEMIOLOGY OF PELLAGRA. REPORT OF PROGRESS.[1]

By Joseph F. Siler, B.S., M.D.,
captain, medical corps, united states army,
and
Philip E. Garrison, A.B., M.D.,
passed assistant surgeon, united states navy.

(From the Laboratory of Tropical Medicine, New York Post-Graduate Medical School.)

Part I.

I. General Plan of the Work. Although the Thompson-McFadden Pellagra Commission will continue its work in the field

[1] From the Thompson-McFadden Pellagra Commission, New York Post-Graduate Medical School and Hospital.

during 1913, it is considered advisable and warranted to set forth at this time in a brief preliminary report certain results of the epidemiological study made in 1912.

It was decided that an intensive study of the disease as it occurred among the population of a limited area would yield more valuable epidemiological information than a more superficial investigation over an extended area, conducted by correspondence and by brief studies in a number of different localities. No such intensive epidemiological study within a small area seems to have been previously undertaken in pellagra, while much data gathered by the more general methods are already available in the literature.

For the collection of data a blank booklet was prepared, covering points considered of possible significance regarding the patients themselves, their families, their residences, and the neighborhood in which they lived. In order to secure these data, each patient was visited one or more times in his or her own home.

In this preliminary report only summarized data will be included. The detailed epidemiological data for each case, and certain more intensive studies made in selected localities within the county, will appear in the report to be issued on completion of the investigation.

The problem immediately before the commission in all its work was the etiology of pellagra and not its symptomatology or therapeutics, except as these subjects might throw light upon the essential nature of the disease. In view of the essential importance of diagnosis in our work, the conservative position was taken that a positive diagnosis would be made only when the characteristic skin lesion was evident or its earlier presence could be definitely ascertained by the testimony of patient and physician, though this requirement might, and ultimately did, exclude from our records cases which in all probability were suffering from pellagra without showing evidences of its cutaneous manifestation.

II. Acknowledgments. It is not practicable to make personal acknowledgment in each case to the many physicians to whom we are under great obligations for their interest and coöperation. The Spartanburg County Medical Society supported us in all our work, and we wish to express our high appreciation of its active part in furthering the investigation. Our only way of approaching patients was through their local physicians, and in no case did we find anything but the most ready coöperation on the part of the physicians, while many actively associated themselves in the work at a considerable expenditure of time and effort. In the more intensive studies in selected localities, which will appear in a future report, opportunity will be given us to make acknowledgment of these special services.

We are indebted to Dr. J. W. Babcock for the privilege of consulting with him from time to time during the work, and for many special services, especially for data concerning pellagrins admitted

to the State Hospital for the Insane from Spartanburg County and for pathologic material collected at autopsies in that institution.

To Dr. J. A. Hayne, secretary and health officer of the South Carolina State Board of Health, we are under obligations for his interest and coöperation in our investigations, and particularly for furnishing from his office vital statistics concerning pellagra.

III. THE TERRITORY SELECTED. Spartanburg County is situated in the northern or Piedmont section of South Carolina. It is forty miles long in a north-south direction, thirty miles from east to west, and contains 762 square miles. Its surface is hilly and broken by a network of small streams and by four small rivers which, with their tributaries, flow across the county in a southeasterly direction, one of these rivers forming the county line on the south. The elevation above sea level at Spartanburg, the county seat, is 875 feet. To the northwest the slope is upward, the northwest corner of the county being situated at the foot of the Blue Ridge Mountain range, while to the south and east the elevation becomes somewhat lower than at Spartanburg, but with no precipitate fall, the whole county thus resting upon a plateau about 700 feet above sea level.

TABLE I.—Population Spartanburg County Census 1910.

		Total.	One year.	One to four years.	Five years.	Six to nine years.	Ten to fourteen years.	Fifteen to seventeen years.	Eighteen to nineteen years.	Twenty years.	Twenty-one to forty-four years.	Forty-five years and over.	Unknown.
Total population	..	83,465	2,733	10,091	2,370	8,852	10,402	5,710	3,878	1,870	26,239	11,161	159
	M.	41,719	1,425	5,167	1,171	4,490	5,270	2,840	1,903	893	12,822	5,652	86
	F.	41,746	1,308	4,924	1,199	4,362	5,132	2,870	1,975	977	13,417	5,509	73
Native white—native parents	..	56,536	1,918	6,842	1,587	5,834	6,742	3,874	2,576	1,254	17,677	8,149	83
	M.	28,472	991	3,526	793	2,969	3,464	1,907	1,280	612	8,820	4,063	47
	F.	28,064	927	3,316	794	2,865	3,278	1,967	1,296	642	8,857	4,086	36
Native white—foreign parents	..	309	5	28	8	29	31	27	8	6	111	54	2
	M.	150	2	15	6	11	12	14	3	2	55	30	
	F.	159	3	13	2	18	19	13	5	4	56	24	2
Foreign-born white	..	203	..	1	..	1	4	6	7	5	98	80	1
	M.	136	..	...	..	..	3	5	6	5	64	53	
	F.	67	..	1	..	1	1	1	1	..	34	27	1
Black	..	21,944	637	2,675	635	2,445	3,018	1,486	1,098	506	6,910	2,467	67
	M.	10,893	335	1,338	299	1,244	1,489	762	530	236	3,289	1,335	36
	F.	11,051	302	1,337	336	1,201	1,529	724	568	270	3,621	1,132	31
Mulatto	..	4,466	172	544	140	543	607	316	189	99	1,441	409	6
	M.	2,065	97	288	73	266	302	152	84	38	593	169	3
	F.	2,401	75	256	67	277	305	164	105	61	848	240	3
Indian	..	4	1	1	..	..	...	..	..	..	1	1	
	M.	1	..	...	..	..	...	..	..	..	...	1	
	F.	3	1	1	..	..	...	..	..	..	1	...	
Chinese	..	2	..	...	..	..	...	..	..	..	1	1	
	M.	2	..	...	..	..	...	..	..	..	1	1	
	F.												
Japanese	..	1	..	...	..	..	...	1					
	M.												
	F.	1	..	...	..	..	...	1					

The annual mean temperature, as recorded at Spartanburg, is 60° F. While the winters are mild, killing frosts are apt to occur from November to March inclusive, and the normal mean temperature for the months of December, January, and February is about 42° F.

The total population of the county is 83,465. Spartanburg, with a population of 17,517, is the only city in the county, the remaining population (65,948) being distributed upon farms, in cotton-mill villages, and among eleven small towns, only two of which have over 1000 inhabitants. The density of population, as a whole, is 109 per square mile; for the rural population (that is, outside of Spartanburg City) it is 86.5. While in South Carolina, as a whole, the negroes form 55 per cent. of the total population, in Spartanburg County the whites predominate numerically in the proportion of somewhat over two whites to one negro—there being a white population of 57,055, and 26,410 negroes—the percentage being 68.4 per cent. whites and 31.6 per cent. negroes. The distribution of the population of the county by race, nativity, age, and sex, according to the United States Census of 1910, is set forth in greater detail in Table I.

The chief industry, and almost the only industry conducted upon a large scale, is that connected with the cotton mills. There are about twenty-eight cotton mills in the county, each mill supporting its mill-village. These mills give employment to approximately 10,000 operatives, representing about 4000 families, and a total mill-village population of about 20,000. As the mill operatives are whites exclusively, it follows that something over 35 per cent. of the white population of the county is found in the mill-villages and is supported by the cotton-mill industry. The mill-village population contains no foreign element, but is drawn altogether from the general native-born population of South Carolina, North Carolina, Tennessee, Kentucky, Georgia, and other nearby States. An effort is made by the mill operators to secure families with the maximum number of individuals capable of employment as operatives. The income of the mill workers ranges from about 75 cents per day to $2 or even more, averaging about $1.25 per day.

The chief agricultural pursuit throughout the county is cotton culture, though in recent years more and more land has been turned over to corn and other grain crops. The average value of farm lands, $36.04[2] per acre, is considerably above that found in many sections of the Southern States.

IV. Prevalence of Pellagra in the County. Altogether, 282 cases of pellagra were studied in detail during the period the commission was working in the county, that is, from June 1 to

[2] United States Census Bureau.

October 15, 1912, and these 282 cases are the basis for the greater part of our study. For the purposes of certain special lines of inquiry, such as the history of pellagra in the county, the geographic distribution of the disease at different periods, the sequence of cases in different local areas, and the possible increase or subsidence of the malady with regard to both frequency and virulence, records were secured of cases known to the physicians of the county, but which had died or moved beyond the county limits. It should be said also that the 282 cases on our list as present in the county in 1912 represent the minimum figures for the county in this period.

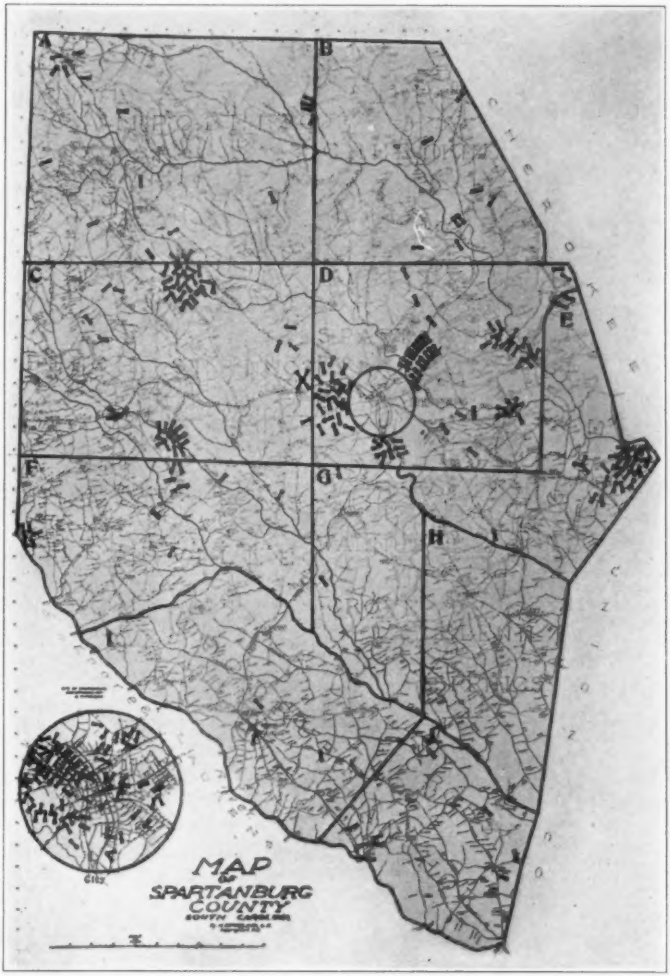
Accepting 282 as the minimum number of cases, we have in Spartanburg County, in 1912, a minimum morbidity rate for the population, as a whole, of 0.35 per cent. or 35 cases of pellagra for each 10,000 of the population.

Including 94 additional cases of which we secured definite knowledge in the county in 1912, but were not able to visit, usually because of their early death or their commitment to the State Hospital, the total number of cases in the county becomes 376, or 44.9 per 10,000 of population. We believe this rate represents very nearly the actual prevalence of pellagra in 1912 in the territory studied.

In view of the fact that this rate is considerably higher than has previously been reported in any single territory of like area, it should be distinctly understood that in no other territory in the United States has so intensive a study been made and pellagrins so thoroughly sought out. Abundant evidence was gathered that the disease is at least equally prevalent in adjoining counties of South Carolina and in certain parts of adjoining States. Furthermore, in Lavinder's extensive compilations of statistics of pellagra in 9 Southern States, 4 States show a greater number of cases than does South Carolina, and 2 States a higher rate per 10,000 of population. Referring to Spartanburg County, Lavinder justly recognizes the exceptional interest displayed by the physicians of the county in the disease, and considers his reports from this county to be exceptionally complete. He was able to get reports of 226 cases up to the beginning of 1912. Our more intensive search, confined to the one county, discovered a total of 398 cases prior to 1912. The difference of 172 cases might be considered surprisingly small were it not for the unquestionable fact that the reports obtained by Lavinder from Spartanburg County were exceptionally complete. There can be no question that the excessive number of cases for Spartanburg County, which appears in both Lavinder's figures and ours, represents more complete returns, and not a greater prevalence of pellagra than is present in other localities from which reports are less satisfactory.

V. Geographical Distribution of Pellagra Within the County. In order first to investigate the possibility of any geographical inequality in the distribution of the disease within

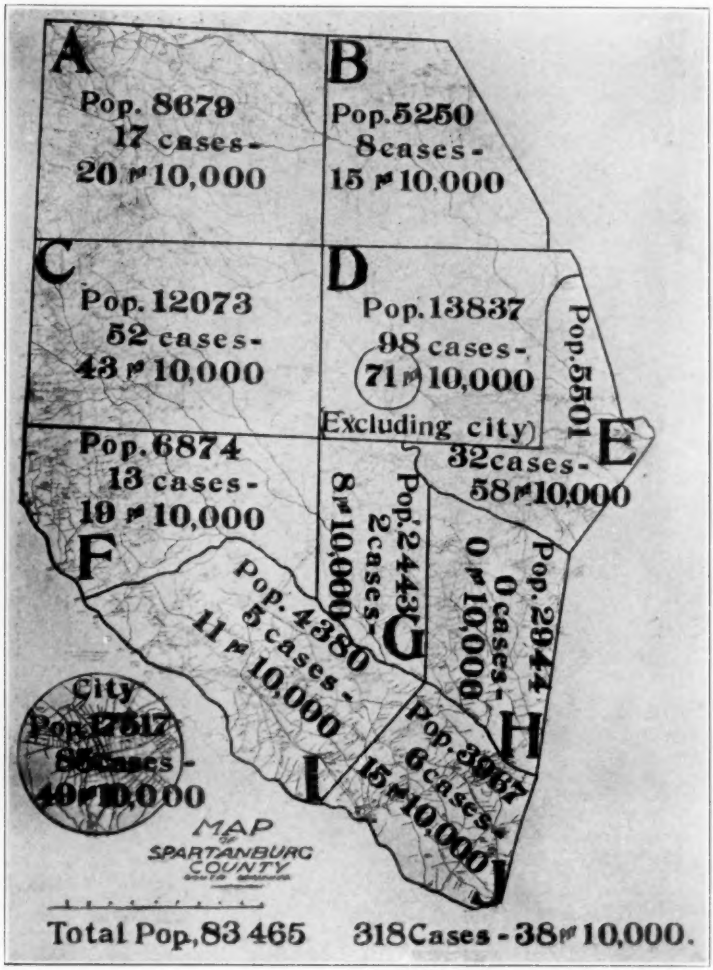
the bounds of the county itself, we have considered the incidence of the disease in each of its ten townships separately.



MAP 1.—Geographical distribution of cases in county and city of Spartanburg in 1912.

By referring to Map 2 it is seen that the township rate per 10,000 of population ranges from no cases in township H to 71 cases per 10,000 of population in township D. It is noteworthy, further, that the three townships C, D, and E, stretching across

the middle of the county, give rates of 43, 71, and 58 cases respectively, or a combined average of 55 per 10,000; while townships



MAP 2.—Distribution of cases by townships with rate per 10,000 of population.

A and B to the north and F, G, H, I, and J to the south give 20, 15, 19, 8, 0, 11, and 15 respectively and a combined average of only 14 per 10,000.

The incidence rate of pellagra within the city of Spartanburg

was 49 per 10,000, considerably lower than the total rate (58) for township D, in which it is situated. The combined average rate of townships C, D, and E, exclusive of Spartanburg City, is 58 against only 14 in the remaining townships.

The population of the county may be still further divided geographically into three parts: (1) the rural population; (2) the mill-village population; (3) the urban population of Spartanburg City. The rural population is found upon the farms and in eleven small towns, one of which has 1880 inhabitants, another 1101, and the remaining nine from 100 to 500 or 600.

Approximately one-fourth of the population of the county live in the cotton-mill villages. There are twenty-eight of these villages in the county, their individual population ranging from about 200 to about 2000. Within the city of Spartanburg there are two

TABLE II.—Rural, Mill-village, and Urban Distribution of Cases by Townships.

Township.	Total population.			Rural population.			Mill-village population.			Excess in mill-village over rural population.
	Population.	Cases of pellagra.	Rate per 10,000.	Population.	Cases of pellagra.	Rate per 10,000.	Population.	Cases of pellagra.	Rate per 10,000.	
A	8,679	17	20	8,179	13	15	500	4	80	65
B	5,250	8	15	4,650	7	13	600	1	17	4
C	12,073	52	43	9,173	17	20	2,900	35	121	101
D[3]	13,837	98	71	7,504	21	28	6,333	77	120	92
E	5,501	32	58	2,751	8	29	2,750	24	87	58
F	6,874	13	19	6,174	8	13	700	5	71	58
G	2,443	2	8	2,443	2	8	0	0	0	
H	2,944	0	0	2,944	0	0	0	0	0	
I	4,380	5	11	3,580	3	8	800	2	25	17
J	3,967	7	18	2,967	4	13	1,000	3	30	23
City	17,517	85	49	(14,567[4])	(43[4])	(29[4])	2,950	42	142	113
County	83,465	319	38	50,365	83	16	18,533	193	104	88

mill-villages, with a joint population of approximately 2950, the remaining 14,567 constituting the city population proper. Such a distribution of the population affords an opportunity to carry farther the analysis of the geographic distribution within the territory of each township by considering separately the prevalence of the disease among the rural, urban, and mill-village population respectively. The results of this analysis are shown in Table II and are graphically displayed by Chart 1.

With one exception, in each of the eight townships with a mill-village population, the prevalence of pellagra among the mill-villages is markedly in excess of its prevalence among the rural population, this excess ranging from 17 per 10,000 in township I to 101 per 10,000 in township C, and reaching even a still higher

[3] Exclusive of city.

[4] City population exclusive of mill-village.

figure (113) in the city of Spartanburg. In the county, as a whole, the mill-village population shows an excess of 50 per 10,000 over the total county average and of 88 per 10,000 over the rural districts alone. The apparent exception presented by township B, in which the excess among the mill-village population was only

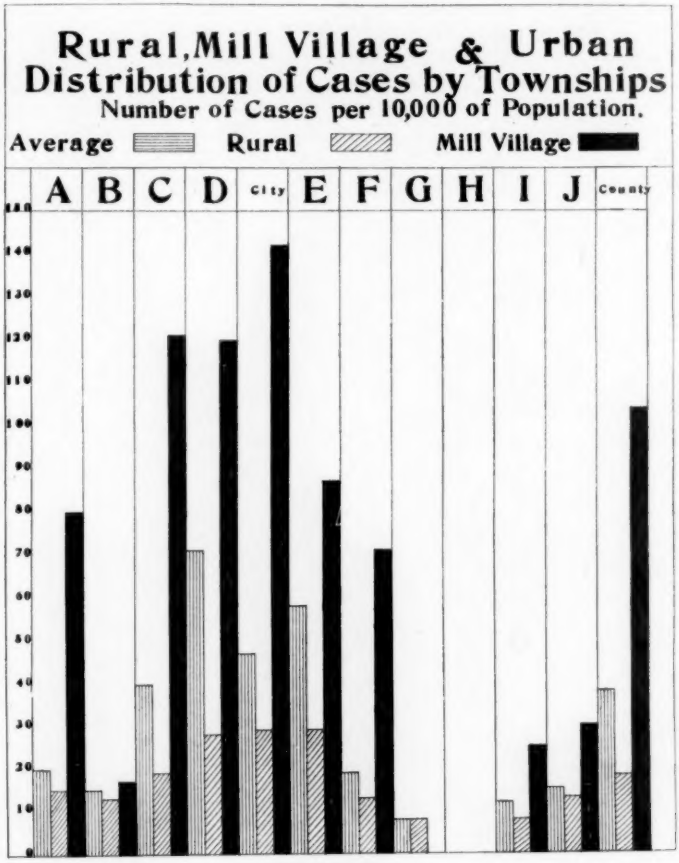


CHART 1.

4, disappears in view of the fact that the one mill in that township had been in operation only a month or so. The rate per 10,000 in the urban population of Spartanburg City, exclusive of the mill-village population of the city, is about equal to the combined rate among the rural population of townships C, D, and E, exclusive

of their mill-village population, while the mill-village population in Spartanburg City (2950) shows 142 per 10,000, the maximum rate in our figures.

Excluding both urban and mill-village population, and considering the rural population alone, we find that the three townships C, D, and E still show a prevalence of pellagra twice that of the remainder of the county, the combined average rural rate for these townships being 24 per 10,000, while for the rural population of the remainder of the county it is only 12.

There are two factors which may possibly offer an explanation of this difference. In the three townships (C, D, and E) showing the excessive rate, with a total population of 19,428, the mill-village population (9833) makes up 50 per cent. of the total population (excluding the city of Spartanburg), while in the remainder of the county, with a total population of 30,937, the mill-villages have a population of only 3600, or 12 per cent. of the whole. It might be supposed that a large mill-village population showing a relatively high prevalence would tend to increase the prevalence of the disease in the surrounding rural districts.

The apparent fact that cases of pellagra are excessively prevalent in the mill-villages where the population is congested, suggests the question whether the congestion of population itself may not have an important influence upon the incidence of the disease. The average density of population for the county as a whole is 109 per square mile, while for the rural districts, exclusive of Spartanburg City, it is 86.5. For the rural population, exclusive of the mill-villages, it is 60.3. In the three townships C, D, and E, which show a striking excess of pellagra among the rural population compared with the rural population of the eight other townships, the density of rural population is 72 per square mile, and in the remaining townships it is only 56, a difference in density of 16 per square mile. While the greater density of population in townships C, D, and E might be a causative factor in the greater prevalence of pellagra in the rural population of those townships, the rate of prevalence does not vary strictly as the relative density of population in each township, though it shows some tendency to do so.

Further evidence that density of population alone is not accountable for the greater prevalence of the disease in mill-villages is found within Spartanburg City itself. There the mill-villages, which are continuous with and an integral part of the city, present a rate of 142 per 10,000, whereas the remainder of the city population, living under approximately the same condition of congestion, gives only 29 per 10,000. Furthermore, the non-mill-village population within the city, with a density which is certainly over 3000 per square mile, shows almost exactly the same prevalence of pellagra per 10,000 as does the strictly rural population of the surrounding township, with only 90 inhabitants per square mile.

These figures would seem to indicate that while congestion of population may play a part in the prevalence of the disease, it alone does not explain the marked inequality of distribution between the mill-villages and the strictly rural population.

VI. Racial Distribution. The distribution of the cases of pellagra in the county between the races presents a second marked inequality. While in South Carolina as a whole the number of whites and negroes is about equal, the negroes being slightly in excess, in Spartanburg County the whites predominate numerically in about the proportion of two to one, the actual figures being, whites 57,055, negroes 26,410 (Table III). The 57,055 whites gave 257 cases of pellagra, or at the rate of 45 per 10,000; the 26,410 negroes gave 25 cases of pellagra, or at the rate of 9.5 per 10,000. In other words, while whites are present in the population in the proportion of two whites to one negro, there are ten white pellagrins to one negro pellagrin.

One fact of fundamental importance in the racial comparison is the practical absence of negroes from the mill population. It follows that a more accurate comparison between the races can be made by comparing the rate of prevalence among negroes with the rate among whites, exclusive of the mill population. We have seen that the rate among the total population, exclusive of the mill-villages, was 18 per 10,000. Subtracting the negro population and the negro pellagrins we have remaining a white population, exclusive of mill-village population, of 38,522, which gave 97 cases of pellagra, or 25.2 per 10,000 against 9.5 per 10,000 among the negroes. In other words, in Spartanburg County the disease appears to be 4.7 times as prevalent among all whites as among negroes, and 2.6 times as prevalent among whites, exclusive of the cotton mill-villages.

Table III.—Distribution of Pellagrins among Whites and Negroes.

	Population.	Cases of pellagra.	Rate per 10,000.	Excess of whites over negroes per 10,000 of population.
All whites	57,055	257	45.0	35.5
Whites outside of mill-villages	38,522	97	25.2	15.7
Negroes	26,410	25	9.5	

The racial incidence was carefully investigated with a view to determine whether the racial variation might be due to failure to discover cases of pellagra among negroes as readily as among whites. The practising physicians throughout the county were questioned as to the comparative prevalence among the negroes in their particular section, and as to its comparative prevalence

in this race in the past. Without exception we were informed that pellagra in negroes was of comparatively infrequent occurrence. In this connection it is well to state that many of these physicians have lived and practised medicine in the same place for many years; that they are personally acquainted with practically the entire population in their particular field of work, both whites and negroes, and that they are in general thoroughly capable of correctly diagnosticating the disease.

The two colored physicians in Spartanburg were closely questioned as to the occurrence of pellagra in their practice at present and in the past, and the cases cited by them are included in our statistics. Furthermore, the information furnished by them relative to the occurrence of cases in the past is in accord with these statistics. When negroes suffering with pellagra were visited a particular effort was made to secure from them information as to the occurrence of the disease in others of their own race. A number of names were secured in this way, and many negroes suspected of having the disease were visited. Many of the planters owning large plantations and having as tenants or laborers a large number of negroes were closely questioned as to the prevalence of the disease, and in some instances canvasses were made. Notwithstanding the efforts made to discover the disease in negroes, it was impossible to find more than twenty-five cases.

It is not believed that the number of cases overlooked could materially alter the relative incidence of nearly five cases in whites to one in negroes, and the racial variation is evidently a real one, though it may possibly be explained in part by the absence of negroes from the mill-village population, which, as we have seen, shows a marked excess of pellagra compared with the remainder of the white population.

TABLE IV.—Racial Distribution of Population and Racial Distribution of Pellagra with Percentages.

State.	Population statistics.[a]				Pellagra statistics.					
	White.		Negro.		White.		Negro.		Pellagra per 10,000 of population.	
	Number.	%	Number.	%	No.	%	No.	%	W.	N.
Virginia	1,389,809	67.4	671,096	32.6	476	76	152	24	3	2
North Carolina	1,500,513	68.0	697,843	31.6	1744	81	407	19	12	6
South Carolina	697,162	44.8	835,843	55.2	1129	71	471	29	16	6
Georgia	1,431,816	54.9	1,176,987	45.1	3127	80	741	20	22	6
Kentucky	2,027,951	88.6	261,656	11.4	442	92	39	8	2	4
Alabama	1,228,832	57.5	908,282	42.4	1138	58	813	42	9	9
Mississippi	786,119	43.7	1,009,487	56.6	1387	55	1156	45	18	11
Louisiana	941,086	56.8	713,874	43.1	338	56	269	44	4	4

[a] Population statistics are those of the Thirteenth Census (1910). The pellagra statistics were compiled from those reported by Lavinder in reprints from Weekly Public Health Reports, No. 106.

Table No. IV is an analysis of population by race, and of the incidence of pellagra by race for eight Southern States. The pellagra statistics in this table were secured by correspondence and, as Lavinder states, are very incomplete.

In Spartanburg County there is a marked difference in racial incidence, but any suggestion that this depends upon the factor of race alone may seriously be questioned. In Table IV it may be seen that the case incidence in the two races in the States of Alabama and Louisiana shows little difference; while in South Carolina and Georgia, and to a less extent in North Carolina, there is a marked preponderance in whites. It is possible that industrial conditions in these States account largely for this variation.

VII. Sex Distribution. The population of the county is divided practically equally between males and females, the females being only 27 in excess. The total male population of 41,719 gave 71 cases of pellagra, or at the rate of 17 per 10,000. The female population gave 211 cases, or at the rate of 50.5 per 10,000 (Table V). In other words, pellagra appears nearly three times more frequently among females than among males. Among the white population alone this proportion between males and females remains about the same, while among the negroes the relative prevalence among males and females is nearly four females to one male. This inequality between the sexes is brought out more strikingly and in greater detail if the figures are analyzed by dividing the population according to age.

Table V.—Distribution by Sex.

		Number in population.[6]	Cases of pellagra.	Rate per 10,000.	Excess per 10,000 among females.
Whites:	Male	28,758	66	22.95	
	Female	28,290	191	67.5	44.5
Negroes:	Male	12,958	5	3.9	
	Female	13,452	20	14.9	11.0
Both races:	Male	41,716	71	17.0	
	Female	41,742	211	50.5	33.5
Total population		83,458[6]	282	33.8	

Table VI.—Age Distribution of Two Hundred and Eighty-two Cases.

Age.	Total population.[7]	Cases of pellagra.	Rate per 10,000.
0 to 5	15,194	21	10.8
6 to 9	8,852	22	24.9
10 to 19	21,860	28	12.8
20 to 44	26,239	158	60.2
45+	11,161	53	47.5

[6] Exclusive of 4 Indians, 2 Chinese, and 1 Japanese.
[7] Exclusive of 159 persons of unknown age.

VIII. Age Distribution. First, considering the prevalence of pellagra according to age, without reference to sex, we find the inequalities shown in Table VI, and graphically presented in Chart 2.

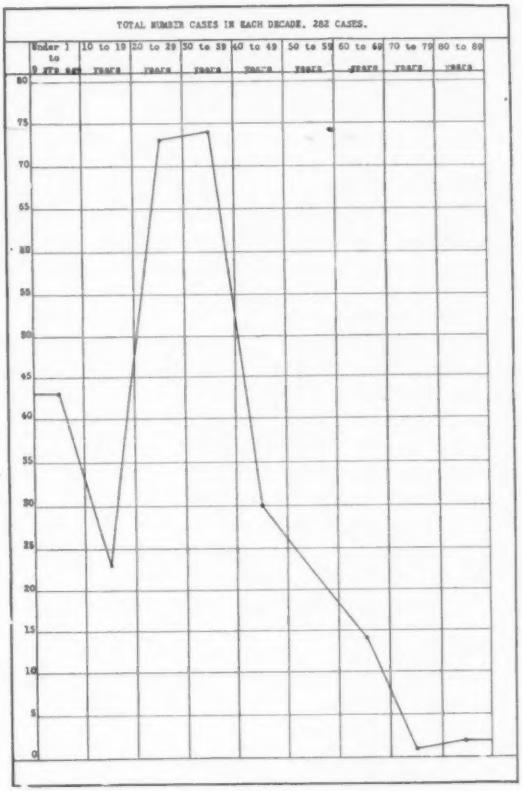


Chart 2.—Total number of cases in each decade.

In this and the following tables dealing with age distribution, in which the prevalence of pellagra is expressed in rate per 10,000 of the population, our age groups are necessarily made to conform to the age statistics for the population of Spartanburg County which could be secured from the United States Census Bureau—namely, the population of the county under six years of age, from six to nine years, ten to nineteen years, twenty to forty-four years, and forty-five years or older. The actual number of cases in these groups means little because of the wide difference in the size of the groups, both with regard to the number of years and the number

of individuals concerned. A striking excess in the rate of prevalence is apparent in the group twenty to forty-four years of age (60.2 per 10,000). The rate among those over forty-four years of age is much higher than in any other group excepting that between twenty and forty-four years. These two groups taken together, that is, the entire population over nineteen years of age compared with the entire population under twenty years of age gives the following result: Twenty years and older, 211 cases, or 56.4 per 10,000; nineteen years and younger, 71 cases, or 15.5 per 10,000.

Dividing the younger group we find that children under ten years of age gave a total of 43 cases, or 17.9 cases per 10,000; those from ten to nineteen years of age gave 28 cases, or 12.8 per 10,000. Furthermore, this higher prevalence for children under ten years is found entirely among the children between the ages of five and ten years who alone gave a rate of 24.9 per 10,000, while the younger group under five years of age gives only 10.8 per 10,000, the lowest rate found in any group.

In Chart 2 the distribution of pellagra by the number of cases in each age group is shown, dividing the cases into age decades. By comparing this chart with Table VI it is evident that the curve would be considerably modified if it expressed the rate per 10,000 of population instead of the actual number of cases in our figures. The prevalence among children under ten years would not be quite so high compared with that in other groups. The relative prevalence in groups over forty years would be higher. The two groups of from twenty to forty years would still show a striking excess over all other decades. It may be said here that the marked fall in the prevalence of pellagra in the groups ten to nineteen years appears throughout our statistics, and will come out strikingly in the consideration of the family distribution of the disease.

IX. Distribution by Age and Sex. It is important to ascertain whether the excessive prevalence of pellagra among females holds in all ages of the population, and whether the excessive prevalence found in certain age groups, notably those from twenty to forty-five years, is to be found among both males and females. Referring to Table VII it may be seen that the excess among females is not the same among all ages of the population, but is confined largely to those between the ages of twenty and forty-four years. Further, under ten years of age males and females show practically the same prevalence of pellagra. From ten to nineteen years the females show a rate a little over twice that of the males; in the large group of from twenty to forty-four years the females show a rate per 10,000 over nine times greater than the male rate. Among the population of forty-five years and over the distribution between the sexes is again nearly equal.

These cases have been further analyzed by decades, with the result as represented graphically by curves in Chart 3. These curves express actual number of cases.

TABLE VII.—Distribution by Age and Sex.

Age.	Sex.	Population.	Number of cases.	Rate per 10,000.	Excess per 10,000.
Under 5	M.	7,763	10	12.9	
	F.	7,431	11	14.8	1.9
5 to 9	M.	4,490	12	27.0	
	F.	4,364	12	27.5	0.5
10 to 19	M.	10,906	7	6.4	
	F.	10,954	16	14.6	8.2
20 to 44	M.	12,822	16	12.5	
	F.	13,417	148	110.3	97.8
Over 44	M.	5,652	25	44.2	
	F.	5,509	27	49.0	4.8

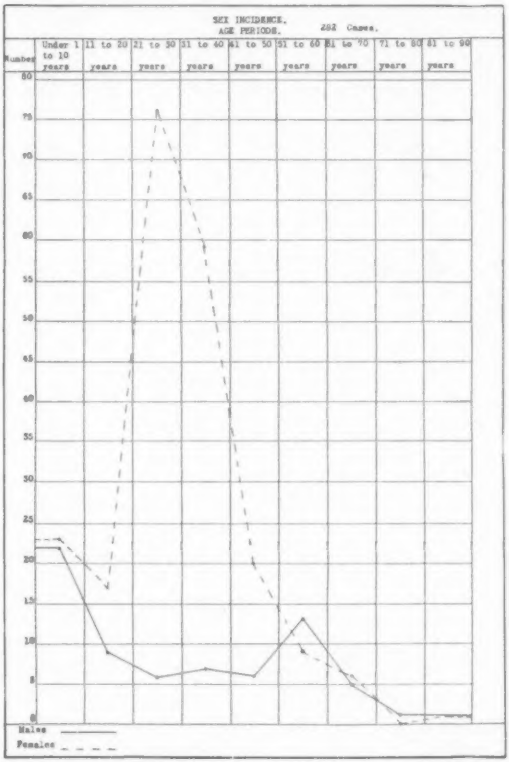


CHART 3.—Distribution by age and sex.

The fall in the curve in the second decade of life is present for both sexes, but is more marked for males. Thereafter the male curve continues to fall, whereas the female curve rises to its highest

point in the following decade (twenty-one to thirty years), drops slightly among women of thirty-one to forty years, then falls abruptly in the next decade (forty-one to fifty years) to a point somewhat below both males and females under ten years. In the decade of from fifty-one to sixty years the female curve continues to fall, and for the first time descends below the male curve, which shows a slight rise. Thereafter the number of cases is small and the two curves fall together.

The data expressed in Table VII and Chart 3 may be summarized as follows: Pellagra appears to be about equally prevalent among males and females under ten years of age and over forty-five years. Males alone show the highest prevalence in children under ten years. Females show a strikingly higher prevalence in the two decades of twenty to thirty and thirty to forty years than does either sex in any other decade.

Table VIII indicates in a general way that both whites and negroes separately show practically the same inequalities in the distribution of pellagra between the sexes in the different age groups, as has been shown in considering both races together. Any closer analysis of the figures for the two races is unsatisfactory, owing to the small number of negro cases in each age group.

TABLE VIII.—Race, Age, and Sex Incidence (Two Hundred and Eighty-two Cases).

Age.	Whites, 257 cases.		Negroes, 25 cases.	
	Male.	Female.	Male.	Female.
Under 1 to 5	9	11	1	0
Under 6 to 10	12	11	0	1
Under 11 to 20	9	14	0	3
Under 21 to 30	4	65	2	11
Under 31 to 40	6	56	1	3
Under 41 to 50	6	20	0	0
Under 51 to 60	14	9	0	0
Under 61 to 70	4	5	1	1
Under 71 to 80	1	0	0	0
Under 81 to 90	1	0	0	1
Totals	66	191	5	20

While it is not proposed to enter into any extensive comparative studies in this preliminary report, there are certain data upon the age and sex distribution of pellagra in this country which present an interesting parallel with the figures for Spartanburg County.

Chart 4 represents by curves based on actual number of cases the age and sex distribution of 164 cases of pellagra, 99 of which were reported by Mizell,[8] of Georgia, and 65 by Tucker,[9] of Virginia.

[8] From paper read at the Second Triennial Meeting of the National Association for the Study of Pellagra, Columbia, S. C., October 3, 1912.

[9] Beverley R. Tucker, M.D., A Discussion of Pellagra, with Remarks on Sixty-six Cases Occurring Outside of Institutions, Old Dominion Jour. Med. and Surg., April, 1911, vol. xii, No. 4.

Chart 5 shows mortality rates for the State of Texas. This chart was furnished to us by Dr. H. K. Beall, of Fort Worth, Texas,[10] who first directed attention to the inequalities of distribution by age in males and in females.

Chart 6 is based upon death reports of pellagra in Alabama between June, 1909, and December, 1912. These reports cover a total of 1148 cases during this period. The data were kindly supplied by Dr. W. H. Sanders and Dr. H. G. Perry, of the Alabama State Board of Health.[11]

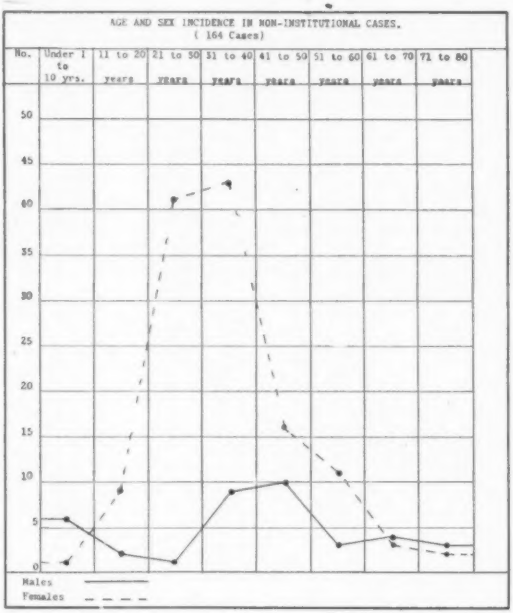


CHART 4.—Age and sex incidence in non-institutional cases.

Chart 7 presents a graphic representation of mortality rates for pellagra in the State of North Carolina for 1911 and 1912. We have to thank the health authorities of North Carolina for the information on which this chart is based.[12]

The data from these five sources are all the statistics available to us at the present time which lend themselves to a comparison with our own figures regarding age and sex distribution. It should be noted that the curves in Chart 4, like our own, are based on morbidity statistics, while Charts 5, 6, and 7 are based upon death reports.

[10] Personal communication. [11] Ibid. [12] Ibid.

A comparison of the four charts shows considerable variation in both the male and the female curves in different age groups.

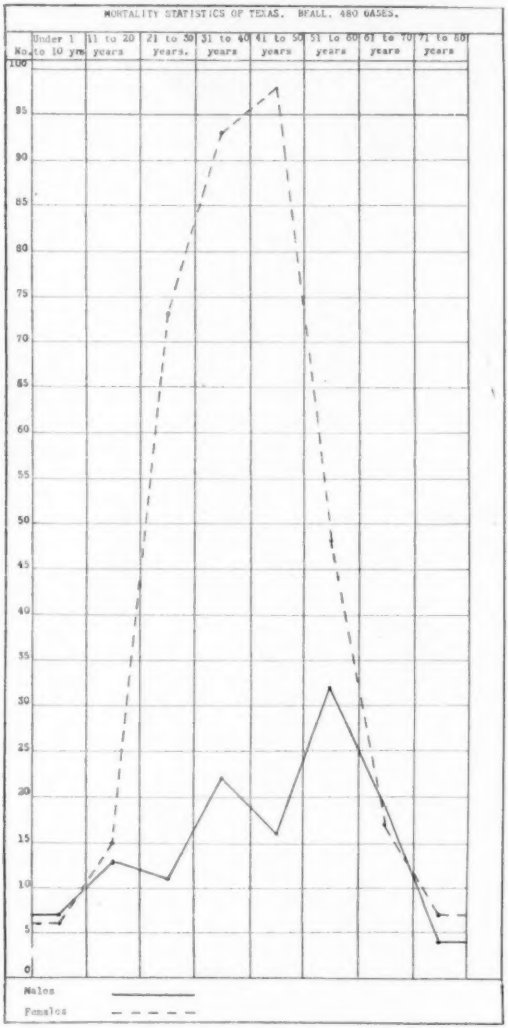


CHART 5.—Mortality statistics of Texas.

They all agree, however, in showing a strikingly excessive prevalence among females of middle age and a comparative equality

of distribution between males and females in childhood and among people of advanced age.

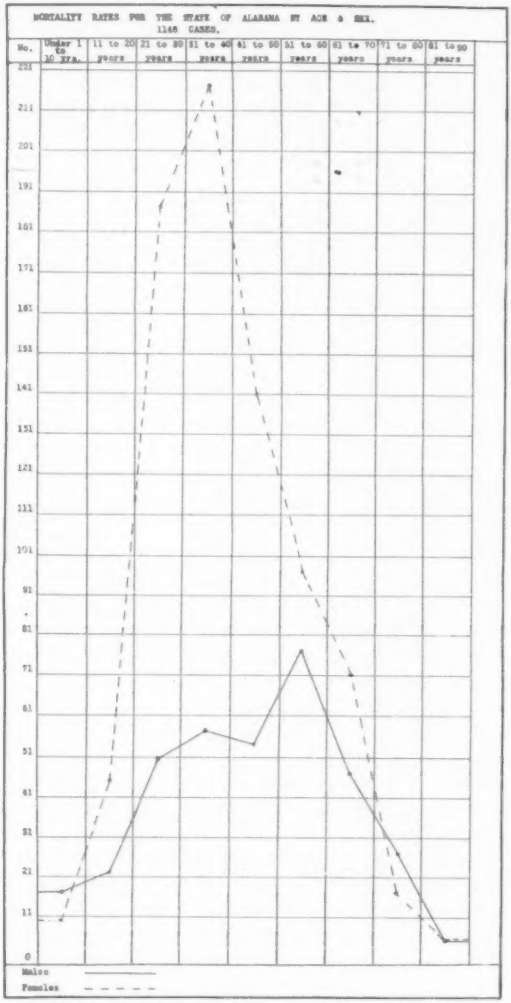


CHART 6.—Mortality rates for the State of Alabama by age and sex.

X. DISTRIBUTION BY OCCUPATION. The consideration of the relationship between occupation and the incidence of pellagra in Spartanburg County resolves itself almost entirely into a dis-

cussion of the relative prevalence of the disease among field laborers, workers in the cotton-mills, and those engaged in housework. Only a few scattered cases gave other employment. The actual data with regard to occupation obtained from 234 cases of pellagra is set forth in Table IX: 110 (47 per cent.) gave housework as their occupation exclusively; 14 others (6 per cent.) gave housework as their chief employment; 16 (6.8 per cent.) were employed in housework part of the time, working the remainder of the time in the mills; 18 (7.7 per cent.) worked alternately about the house and in the fields. Thus a total of 158 (67.5 per cent.) of

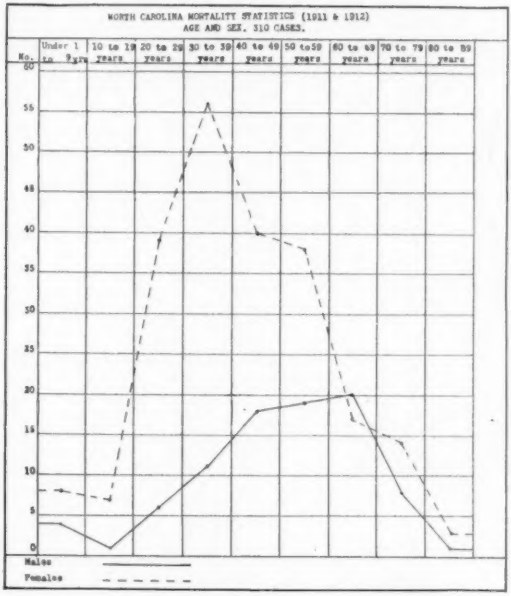


CHART 7.—North Carolina mortality statistics during 1911 and 1912.

the 234 cases, were employed in household work within the home for at least a fair portion of their time. These figures mean little more than what has already been shown by the age and sex distribution of the disease—namely, that it is excessively prevalent among adult females the great majority of whom are employed in housework. There is one further indication, however, that pellagra has a much higher prevalence among the adult females of the mill-villages who are occupied as housewives than among those who work in the mills, and this point may be more closely examined by considering the occupational distribution of pellagra in the mill population alone.

TABLE IX.—Distribution of Two Hundred and Thirty-four Cases of Pellagra by Occupations.

Occupation.	Males.	Females.	Both sexes.	Per cent. of total.
Farmers	14	..	14	6.0
Field work and housework	..	18	18	7.7
Mill work exclusively	21	20	41	17.5
Mill work, some housework	..	16	16	6.8
Housework exclusively	..	110	110	47.0
Housework chiefly, some mill work	..	14	14	6.0
Scattering[13]	9	3	12	5.1
No occupation	2	7	9	3.8

There are available for this study 121 cases of pellagra of working age residing in mill-villages. Of these, 24 were males and 97 females; 21 (87.5 per cent.) of the 24 males were mill operatives. Of the 97 females, 12 (only 12.37 per cent.) worked in the mills exclusively; 16 additional female cases worked in the mills the greater part of the time, making a total of 28 female mill workers (or 28.86 per cent.) of the 97.

46, or very nearly half (47.42 per cent.) of the 97 women did no mill work, devoting themselves to housework exclusively; while 14 others were chiefly employed in housework, going to the mills only occasionally, making a total of 60 houseworkers, or 68.85 per cent. of the female pellagrins of working age living in the mill-villages.

We have no actual statistics regarding the proportion of males and females among mill operatives. It would seem perfectly safe to say that there are at least as many females as males, the probabilities being that they are in a considerable majority. As a rule, female operatives are preferred by the mill operators. Assuming equality between the sexes among mill workers, as may surely be done with safety, the data above presented has a highly important significance, in that it shows a nearly equal prevalence of pellagra in males and females who are employed in the mills as operatives—namely, 17 males and 12 females—if we include only those women doing mill work exclusively, and 28 females if we include the 16 who gave mill work as their chief but not exclusive occupation. This comparative equality as regards the prevalence of pellagra between the sexes among mill operatives is in striking contrast to the inequality between adult males and females in our sex statistics for the population as a whole, and is very closely in harmony with what is known of sex distribution in institutional cases where a difference in prevalence between the sexes is absent or slight.

In comparing the mill-village population with the rural popu-

[13] Two patients were merchants, two dressmakers, one was employed in each of the following occupations: insurance, student, carpenter, butcher, clerk, railroad fireman, day laborer, school teacher.

lation as regards the prevalence of pellagra, we found the disease nearly seven times as prevalent in the mill-villages as in the rural districts, the actual rate of prevalence in the mill population being 104 per 10,000. Out of the total mill-village population of approximately 19,000, about one-half, or 9500, are actually employed in the mills. Among these mill operatives we have 57 cases of pellagra, including those patients who gave mill work as their chief but not exclusive occupation. These 57 cases give a rate of 60 per 10,000 among actual mill operatives, against 104 per 10,000 for the total mill-village population. The 41 patients giving a history of mill work exclusively present a rate of only 43.2 per 10,000. These figures seem to indicate that the excessive prevalence of pellagra in the mill-village population is not found among those who actually work in the mills, but among the women engaged in the day about the houses, and the children who are at home with them.

While children under working age do not form a part of an occupational study, it may well be noted in this connection that 30 mill-village children under ten years of age had pellagra. These cases added to the 60 adult females in the mill-villages engaged in housework exclusively make a total of 90 cases of pellagra in the mill-village population of the county, which by occupation were about the dwellings during the day, against 57 cases among the population engaged in mill work. The 57 operatives give a rate of 60 per 10,000 for the half of the mill-village population which works in the mills. The 90 houseworkers and children give a rate of 94.7 per 10,000.

XI. Distribution of Cases in Families. 316 cases of pellagra are available in our data for a study of family and household relationships, and these cases represent 223 families, an average of 1.42 cases of pellagra per family. An analysis of the family distribution is given in Table X. About half the total number of cases occurred singly in families, and about one-fourth of the total number occurred two to a family. Of the total number of families with pellagra (223) nearly three-fourths (160) had but one case, and nearly one-fifth gave but two cases. Nearly one-tenth of the families gave 3, 4, or 5 cases.

In view of the inequalities found in the prevalence of pellagra in the two sexes, and at different ages, an attempt has been made to discover whether the sex and age distribution differs among cases which occur singly in families from the distribution among cases occurring two or more to a family—in other words, to determine whether cases developing singly in families are apt to be of any particular age or sex, and to differ in these respects from multiple family cases. For this purpose, 294 cases for which age and sex statistics are available have been charted (Chart 8) by age periods of five years each—cases occurring singly in families being represented by a solid line, and cases occurring two or more to

a family by a broken line. The upper two curves represent male cases alone; the middle curves, females alone; and the lower curves, the two sexes combined. The curves represent the percentage of the total 294 cases and not the actual number of cases in each group. Accordingly, the solid line and the broken line would coincide wherever the same condition of distribution exists between single cases and cases occurring two or more to the family.

TABLE X.—Distribution of Pellagra in Families.

	Number of families.	Number of cases.	Percentage of total number of families.	Percentage of total number of cases.
One case to one family . .	160	160	71.7	50.6
Two cases to one family . .	42	84	18.8	26.6
Three cases to one family .	14	42	6.3	13.3
Four cases to one family .	5	20	2.3	6.3
Five cases to one family . .	2	10	0.9	3.2
Total with more than one case to one family . .	63	156	28.3	49.4

The most striking inequality between the two curves is present among children under ten years of age. Not only is the curve for multiple family cases higher than the curve for single cases in both males and females, but for males alone and for the combined sexes it goes higher among children under ten years than in any other age group. In this respect it presents a striking contrast to the curve for single family cases and to our general age distribution. It is remarkable, further, that this excessive prevalence in multiple family cases is much greater among males than among females, especially in the younger group of children under six years of age. Among cases occurring singly in families the curve never goes above 5 per cent. in any age group under twenty years among males, females, or the sexes combined. These facts seem to show that where single cases of pellagra occur in families it is rarely children under ten years who are the ones attacked. Among families with more than one case, however, children under ten years form a higher percentage of the cases than does any other decade. Among families with but one case it would appear that the two decades from twenty-one to forty years give the great majority of cases among females, while among males more single cases occur among those over fifty years.

When considering the subject of age and sex distribution, attention was called to the striking fall of prevalence apparent among persons from eleven to twenty years old. Inspection of the curves in Chart 8 shows this fall to be confined almost entirely to cases which occur two or more per family, the indication being that

isolated cases in families are as apt to arise among individuals from eleven to twenty years of age as among children under eleven years.

The Age and Sex Distribution of Pellagra in Families with a single case and in Families with two or more cases

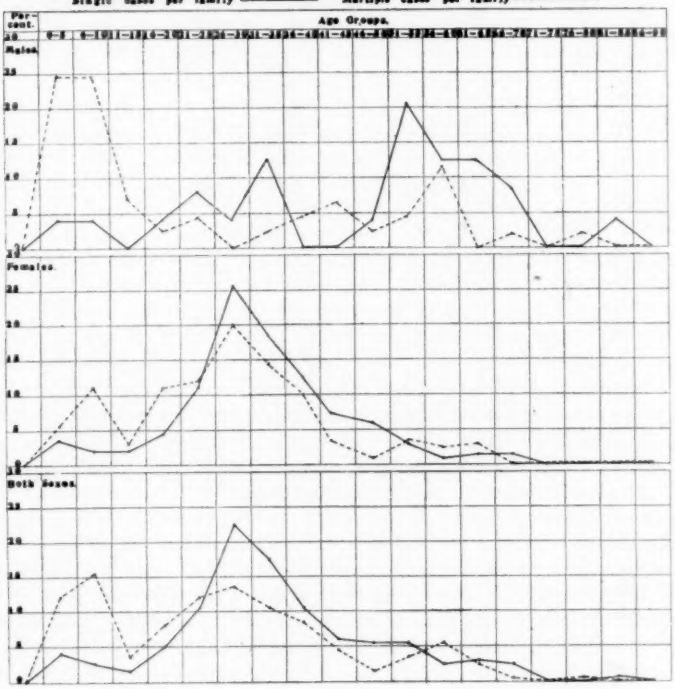


CHART 8.

It is not considered advisable to proceed farther at the present time in an analysis of the family relationships of pellagrins. Such further investigation would carry us into a study of each individual family, the actual sequence of cases in each family, and, furthermore, would introduce the whole subject of household association and its significance with regard to the family relationship. As yet our data are not sufficiently complete to make such a study in a satisfactory manner. It may be said, however, that while there is some evidence in our preliminary investigation that family relationship *per se* does seem to have some influence upon the incidence of pellagra, there are stronger indications that household association is a more important factor in the distribution of the disease.

(To be concluded.)

THE DIAGNOSIS OF INFLAMMATION IN THE ANTERIOR OCULAR SEGMENT.[1]

By B. F. Baer, Jr., B.S., M.D.,

ASSISTANT OPHTHALMIC SURGEON, EYE DISPENSARY OF THE HOSPITAL OF THE UNIVERSITY OF PENNSYLVANIA, PHILADELPHIA.

The ability to diagnosticate ocular inflammations is of the utmost importance. Delay in instituting treatment may result in irreparable damage to the ocular tissues, and faulty diagnosis may be followed by treatment fatal to vision. Far too often has the sight of a glaucomatous eye been destroyed by the instillation of atropine under the impression that the disease was iritis.

The purpose of this paper is to facilitate diagnosis by calling attention to the signs indicative of the forms of inflammation likely to be encountered in one's daily work.

Inflammations in the anterior ocular segment may be divided into those of the conjunctiva, the cornea, the iris and ciliary body, glaucoma, and inflammations consecutive to violence.

These inflammations have as the most noticeable feature vascular injection. The vessels participating in this injection are either the posterior conjunctival or the anterior ciliary, and depending upon which of these systems is congested we speak of conjunctival or ciliary injection. Conjunctival injection appears as a superficial network of bright red bloodvessels whose position in the conjunctiva is demonstrated by the fact that movement of this membrane causes a corresponding change in position of these vessels. The vessels can be individually distinguished. This is the injection of conjunctivitis. Ciliary injection appears as a dark red or violaceous zone surrounding the cornea, and its position beneath the conjunctiva is demonstrated by the fact that movement of this membrane does not result in a change of position of this zone. Instead of the individual vessels being distinguishable there is a general diffuse redness. This is the characteristic injection of inflammation of the cornea and iris and ciliary body.

Acute contagious conjunctivitis, or pink eye, is the classic type of conjunctivitis. The symptoms, conjunctival injection, lachrymation, photophobia, and mucopurulent discharge together with some burning and itching of the eyes, are those which exist with slight variations in all forms of conjunctivitis. This form of conjunctivitis responds readily to argyrol and boric acid flushings, and is of interest because of its epidemic tendency, one case frequently being responsible for the spread of the disease through an entire school.

[1] Read before the staff of the Physicians' and Surgeons' Hospital, Wilmington, Delaware, October 7, 1912.

Gonorrheal conjunctivitis, of which there is the adult form and that seen in the newborn, is a destructive disease which beginning as conjunctivitis may rapidly extend to the adjacent tissues, producing in a few days even a purulent panophthalmitis. Thanks to Crede this is now a rare disease in the newborn. By the use of silver nitrate instillations in the eyes of all infants born in the Leipsic Lying-in Asylum, Crede reduced the percentage of ophthalmia neonatorum from 10.8 to between 0.1 and 0.2. The benefits obtained from this treatment are so immeasurable that Crede's prophylactic treatment cannot be too often retold. During the first bath the eyes are cleansed with pure water—not that of the bath—and cotton, and one drop of a 2 per cent. silver nitrate solution instilled into each eye. Since ophthalmia neonatorum may originate from other than gonorrheal organisms, this treatment should be used in all newborn infants. Most of the cases of gonorrheal conjunctivitis we now see belong to the adult form, and occur in patients suffering with gonorrheal urethritis or in those who have become infected through using the towels and linen previously used by gonorrheal subjects. After an incubation period varying from two or forty-eight hours the lids become red and swollen, often to such a degree that they can only with difficulty be separated. The tarsal and ocular conjunctivæ are intensely edematous, and have a rough, granular appearance. The swelling of the ocular conjunctiva ends abruptly at the margin of the cornea, and the cornea appears as if lying in a pit. The secretion during this stage is serous and tinged with blood, having much the appearance of beef juice. After two or three days the secretion becomes purulent, and the chemosis and swelling begin to subside. The most dreaded complication is involvement of the cornea, resulting in ulceration and even panophthalmitis. Treatment must first be directed toward the prevention of the infection of the second eye if it is still uninfected. This is done by hermetically sealing this eye with a watch-glass strapped down with adhesive tape. Treatment of the infected eye consists of cold compresses, instillations of argyrol, and frequent cleansing of the eyes with 1 to 5000 potassium permanganate solution. As soon as pus begins to flow from the eyes and the swelling in the lids is sufficiently reduced to allow of their eversion the tarsal conjunctiva must be painted with a 2 per cent. silver nitrate solution twice daily, care being taken that the solution does not come in contact with the cornea. When the cornea appears cloudy or its surface dulled, hot compresses must be substituted for the cold, in the effort to stimulate corneal nutrition. Gonorrheal opthalmia is so contagious that the most rigid isolation of these patients is necessary. While it is rare to find an infection of the conjunctiva showing such violent inflammation as that seen following a gonococcal invasion, other organisms will sometimes give rise to a condition which in the early stages resembles

gonorrheal conjunctivitis. A resort to microscopic examination will differentiate these conditions, as the gonococcus is present if the inflammation is due to its influence.

In scrofulous children, phlyctenular conjunctivitis is a common disorder. This disease shows, in addition to the usual signs of conjunctivitis, small reddish tumefactions, about the size of a split pea or smaller, around the margin of the cornea, and often in the cornea itself. After two or three days these phlyctenules break down into small ulcers, which rapidly heal over, only to be succeeded by new crops, the process continuing over weeks and months. If, in the cornea, they leave after healing permanent opacities, which when central materially interfere with vision. Treatment of phlyctenular conjunctivitis, or if the cornea is involved, phlyctenular keratitis, should aim toward the upbuilding of the general system. The local treatment consists of atropine for its sedative effect, frequent cleansing of the eyes, and protection of the eye from light by the use of dark glasses.

Inflammation of the cornea, keratitis, shows in addition to ciliary injection some change in the polish and evenness of the surface, or some loss in the transparency of the cornea. Superficial lesions of the cornea, for example, a phlyctenular or other ulcer, appear as spots in which the lustre and evenness of the cornea are lost, the area being somewhat depressed below the general surface. If fluorescin is dropped into such an eye the spots stain a bright green. A whitish halo of corneal infiltration surrounds each of these areas. Change in the transparency of the cornea is due to change in the deeper layers, such, for example, as is seen in interstitial keratitis. Were one to examine the eyes of the little victim of congenital syphilis, with his large forehead, deeply lined face, and weeping eyes, gray or whitish spots deeply situated in the generally cloudy cornea could be seen; or if these spots were so numerous as to have become confluent, the cornea would have a general ground-glass appearance, often so dense as to obscure the underlying iris. Closer examination of these eyes would show vascular twigs, branching broom-like, appearing suddenly from beneath the corneal limbus, and penetrating toward the pupillary area.

In iritis and inflammation of the ciliary body, cyclitis, the characteristic changes are naturally in these tissues, and, owing to their location, changes occur also in the aqueous and anterior chamber. The iris is hyperemic and swollen, and as a result the pupil is small and non-responsive to light. Inflammatory exudates in the iris tissue result in change of color of the iris, readily observed by comparison with its fellow iris. The aqueous is turbid, and frequently deposits are seen on the posterior surface of the cornea as small dark-colored points over the lower half of Descemet's membrane. The pupillary margin of the iris, where it lies in con-

tact with the anterior lens capsule, becomes glued to this structure at various points. These points of attachment, posterior synechia, upon dilatation of the pupil with atropin, become evident, as a result of which the pupil appears irregularly round, the attached points not yielding to the drug action. Aside from traumatism, iritis generally arises as a result of some constitutional condition, of which syphilis and rheumatism are the most frequent. Tuberculosis, gonorrhea, metabolic disturbances, and acute infectious fevers are responsible for a certain proportion of the cases. Treatment must therefore be directed against the constitutional condition. Locally, antiphlogistic measures and atropine should be employed. Potassium iodide and mercury facilitate absorption of the inflammatory exudates.

When there is a disproportion between the inflow and outflow of the circulating fluids in the eye, the inflow being in excess, there is a rise in intra-ocular tension. Such a condition is called glaucoma. The symptoms of glaucoma, including cupping of the nerve head and loss of sight, originate as a consequence of this elevation of tension.

Beginning with gradual elevation of tension, lasting over months, during which time the patient has attacks of cloudy vision, dull headache, sees colored rings around the street lights (halo vision), the attacks becoming more frequent and severe with the progress of the disease, this prodromal stage finally culminates in an acute glaucomatous attack, with violent pain in the eye, face, and jaws, vomiting and fever, sudden and almost complete loss of vision, injection of the eye, and a dilatation and rigidity of the pupil. The tension at this time, if tested, would be found to be extremely high. With these symptoms the trouble can be easily recognized, yet these attacks are sometimes mistaken for hemicrania. Such an attack may result in blindness in a few hours or, as more usually happens, the tension gradually goes down and the irritating signs disappear, leaving as a permanent aftermath slight reduction in visual acuity. A few weeks later, with a gradual elevation of the tension, a second attack supervenes, only to be succeeded by future attacks, each of which leaves the eye more permanently impaired, until finally it is stony hard, totally blind, and the seat of constant severe pain. This so-called acute inflammatory glaucoma is a rare condition.

In chronic inflammatory glaucoma, the more usual form, the eye passes gradually from the prodromal stage into a glaucomatous condition, with increasing redness of the globe, increasing dilatation of the pupil, and the iris slowly becoming atrophic. The tension rises slowly, but persistently, and at the same time the visual acuity recedes. The attack is not so severe as occurs in acute glaucoma, although the ultimate result of the disease is the same in both forms. Chronic inflammatory glaucoma is sometimes mistaken

for iritis, and unfortunately treated with atropine. One of my preceptors in opthalmology not long ago told me of the following experience. A medical practioner brought a patient to him who for the previous three weeks had been using, with no apparent benefit, medicines prescribed for conjunctivitis. The inflammation happened to be iritis, and promptly subsided under proper treatment. The mental process at work in the mind of this practitioner must have been as follows: if a case when treated as conjunctivitis does not get well, try atropine, it may be iritis; for not long after he called in the same consultant to ascertain why, in another case, atropine was not yielding the desired result. This case was, unfortunately, chronic inflammatory glaucoma, and when first seen by the oculist the sight was entirely obliterated. I have encountered three cases of a similar nature, in each of which the sight was lost.

The uveal tract (iris, cilia y body, and choroid) supplies the ocular fluids, and Schlemm's canal is the channel through which the excess is carried off. In order to reach Schlemm's canal these fluids must first filter through the ligamentum pectinatum, a layer of loose cellular tissue which extends from the root of the iris forward to the point of junction of the sclera and cornea in the filtration angle. Dilatation of the pupil allows the iris to fill in this angle and overlie the ligamentum pectinatum, thus impeding the outflow of the ocular fluids. This happens in glaucoma, and if in such a condition further dilatation of the pupil is induced, as by the instillation of atropine, the filtration of the ocular fluids is further impeded.

Still a third form of glaucoma, chronic non-inflammatory, has, as can be inferred from its name, little or no inflammatory reaction, the diagnosis depending upon the ophthalmoscopic findings and the behavior of the visual fields.

The types of glaucoma above described are classified as primary in contradistinction to secondary glaucoma, in which either during or as the result of some other ocular disease the intra-ocular tension rises above the normal. Secondary glaucoma is sometimes observed in the treatment of iritis, where, as a result of the dilatation of the pupil, the iris is pushed into the filtration angle, thus interfering with the outflow of the ocular fluids. After a severe iridocyclitis, with the production of a total annular synechia, or a pupillary membrane, the posterior chamber is completely closed from the anterior, in this way blocking the natural lymph flow.

Violence to the eye may result in inflammation to one or more of the ocular tissues, and will show the type of inflammation characteristic to the tissues involved. Traumatism may result in a rupture of the ocular tunics, with or without coincident prolapse of the contents of the eye. Stab wounds and the penetration of foreign bodies also causing breaks in the ocular tunics; all such injuries

bring up the question of a retained foreign body, and where the history of the accident does not settle this point, efforts should be made to locate a foreign body by means of direct, ophthalmoscopic, and *x*-ray examinations. If located, efforts must be made to remove the foreign body. The character of inflammation generally set up as a result of penetrating wounds of the globe and retained foreign bodies is iridocyclitis. Iridocyclitis of traumatic origin is responsible for the majority of cases of sympathetic inflammation in the second eye. Patients suffering from an iridocyclitis of the above-mentioned character must therefore be carefully watched for the beginning signs of sympathetic inflammation, which are photophobia, lacrymation, ciliary injection, deposits on the posterior corneal surface, posterior synechia, vitreous opacities, and hyperemia of the retina and optic nerve head. With the development of these signs in the uninjured eye removal of the injured eye is imperative.

The subjective symptoms of the inflammatory diseases in the anterior ocular segment yield little definite information except in glaucoma.

It is seldom that one of the ocular structures is the seat of disease without some involvement in the adjacent tissues. And so involved may this participation of two or more tissues in one inflammatory process become that even the skilled oculist has difficulty in properly differentiating the existing signs. Conjunctival injection denotes conjunctivitis, and ciliary injection shows the process to be of deeper origin. Ciliary injection occurring in the course of conjunctivitis shows an extension of the inflammation to the deeper tissues. Conjunctival injection often exists simultaneously with ciliary injection in severe inflammations, but in these cases the significant sign is the ciliary injection, and it is incumbent upon the physician to ascertain the cause for its presence, and not regard the case as merely a conjunctival inflammation. In iritis the pupil is contracted, whereas in glaucoma it is dilated. In conclusion, let me once again warn against the instillation of atropine or other mydriatic into an inflamed eye until the possibility of glaucoma as a cause of the symptoms is eliminated.

EPIDIDYMITIS DUE TO THE COLON BACILLUS.

By Walter S. Reynolds, M.D.,

CHIEF OF THE CLINIC OF GENITO-URINARY SURGERY IN THE MEDICAL DEPARTMENT OF COLUMBIA UNIVERSITY, NEW YORK.

The subject of colon bacillus infection of the urinary tract has for some time been receiving considerable attention, and as a result cases have been reported where this group of organisms

was found in pure culture accompanying pathological conditions in all these structures. It has also been shown that they may be frequently found in the urine where no pathological condition is present. The frequency, therefore, with which these organisms may be found present under both normal and abnormal conditions leads one to believe that possibly they may be the cause of disturbances in these organs much more frequently than has been supposed. Aside from cystitis, where for some time it has been known that the inflammation, in the majority of instances, was due to the colon bacillus, these organisms did not appear to play an important role in diseases of the urinary organs, though there would seem to be no reason why other organs should not be equally liable to involvement under similar conditions. I have felt satisfied for some time that cases of chronic gonorrheal urethritis were prolonged by the presence of these organisms, and while they have not seemed to be especially virulent in these cases, it is often difficult to get rid of them; but when this is accomplished, I have found that the chronic urethritis improved.

I was lately asked by a friend to examine some slides of secretion from the genital organs of a woman who complained of a discharge which stained her underclothes and who was found to have a Bartholinitis. There was a great profusion of colon bacilli, but no other organism could be found. Her husband had probably not been infected, as he made no complaint. I have at present under treatment a young man who one week after intercourse noticed a slight discharge from the urethra unaccompanied by any marked urinary symptoms, except some slight increase in frequency of urination, and where the only organism to be found in the discharge was a profusion of those resembling colon bacilli. Massage of the prostate showed the same organisms in the expressed fluid. Naturally the question arising in this case is whether he has acquired the infection in intercourse or was the infection derived from his own system?

While it seems reasonably certain that these organisms, though perhaps under ordinary circumstances they are not very virulent, may quite frequently be the cause of inflammatory conditions of the urinary tract, still there are few cases recorded of epididymitis as a result of colon bacillus infection, a complication of frequent occurrence in other infections, such as gonorrhea and tuberculosis.

The manner in which infection of the urinary tract is brought about is still a matter of discussion, and it is also unsettled as to whether it is necessary that a lesion of the intestinal tract be present before the organisms are liberated. In epididymitis occurring with some other diseases we see cases where it seems clear that the epididymis becomes involved by direct extension through the ejaculatory ducts from the posterior urethra or from the prostate. Whether it would be possible for infection to take place in this

way, supposing no inflammatory lesion to be present about the opening of these ducts, is questionable, though we find reports of cases of epididymitis where colon bacilli were found in the urine, but no symptoms of urethritis were present. In such cases the possibility of infection taking place in such manner must, of course, be considered. It is to be remembered, however, that a mild posterior urethritis or prostitis may give rise to no marked symptoms, and it might be possible in some cases to overlook it unless a careful examination was made.

Dr. George G. Smith[1] reports a case of epididymitis following typhoid fever where colon bacilli were found in the urine and culture of the fluid from the epididymis after operation showed pure culture of typhoid bacilli. Epididymitis following typhoid fever is a complication somewhat rarely seen, and the majority of works do not mention it at all. There is considerable doubt as to whether it is an orchitis or epididymitis. Both Osler and Pepper speak of it as an orchitis, and Osler says, it occurs with a mild catarrhal urethritis. In Sajous' *Annual* there is no mention of orchitis or epididymitis in the article on typhoid fever. It does, however, state that "Pus is not infrequently found in the urine of typhoid patients," and says, "It may arise from cystitis or pyelitis." In the same article, under pathological anatomy, we find: "Catarrh of the bladder is sometimes met with, and may be brought about by the careless use of the catheter for retention." So it would seem that uncertainty exists not only as to whether it is an epididymitis or an orchitis, but as to the effect of the typhoid bacilli on the urinary tract as well. It is unfortunate that so many loose statements are made in medical works that it is often difficult to get at the real facts. In Smith's case the operation showed that it was the epididymis which was affected, and the testicle was only slightly if at all involved.

The condition of the intestinal tract in these cases might, with little doubt, afford an open gateway for the escape of the organisms into the circulation, and unless it can be shown that a posterior urethritis is present it would seem more reasonable to suppose that infection takes place in that way notwithstanding the fact that the organisms may be found in the urine, rather than to believe that they pass into the epididymis through the openings of the ejaculatory ducts. Generally speaking, an epididymitis seems to be preceded by a posterior urethritis or prostitis, while orchitis results from blood infection.

In inflammation of the testicle following mumps the infection is undoubtedly carried by the blood stream. Nothing is known as to the nature of the infection, as these cases seldom come to autopsy, and are never operated upon. In like manner in syphilis the testicle is the part first involved.

[1] Trans. Amer. Urolog. Assoc., 1912.

It is of interest to observe that since operations for gonorrheal epididymitis have become more frequent, cultivation of the fluid obtained from the inflamed part, while frequently showing the gonococcus on cultivation, in some instances no organisms can be found. It is assumed, however, that the gonococcus is the cause of the inflammation, since it follows a gonorrheal urethritis. Hagner in several articles on the subject has an interesting series of cases showing the results of such examinations after operations.

In tuberculosis it is the epididymis which nearly always is involved before the testicle becomes affected. But in tuberculosis it is believed by many that the epididymis is always a secondary involvement to tuberculosis elsewhere, and is frequently dependent on tuberculosis of some of the urinary organs.

CASE.—J. D. I., an unmarried man, aged thirty-seven years, was referred to me on May 7, 1912. The previous day he had begun to have frequency of urination accompanied by much pain and tenesmus. The only cause which could be assigned for the trouble was that he had partaken freely of asparagus for dinner the night before, and had also had a cocktail or two. He never had urethritis, but had been treated for catarrhal prostatitis some two years before, which had been relieved by massage and instillations of silver nitrate, otherwise he had been perfectly well.

Examination showed no inflammatory condition of the meatus or urethra, and no discharge was present. Urine passed in two glasses was equally cloudy—in both the cloudiness was due to pus. The urine was not high colored, as he had begun to drink water freely as soon as the frequency of urination had commenced. Examination of the prostate did not reveal any marked prostatic trouble. His tongue was heavily coated, but there was no apparent intestinal disturbance. A diagnosis of cystitis was made, and he was ordered active catharsis, hot rectal irrigations, suppositories of morphine at night, and an alkaline diuretic. After a few days of this treatment the pain, tenesmus, and frequency of urination had so much subsided that he was given bladder irrigations of a solution of boric acid.

A specimen of urine obtained from the bladder after washing with the boric acid solution was examined by Dr. Peter Irving, and showed "many pus cells, epithelium in fair amount, which looks like that from the kidney pelvis, and organisms, probably colon bacilli." A culture was made by Dr. D. S. Jessup, which proved them to be unquestionably colon bacilli. On this finding he was requested to prepare a vaccine which could be used if necessary. Dr. Irving reported the urine to contain no indican or indol-acetic acid, although a pure culture of colon bacilli was present.

On the tenth day after I first saw him he began to have some pain and tenderness in the left cord and epididymis, with some return of the urinary symptoms. Cold lead and opium solution

with an ice-bag and support for the testicle did not prevent the epididymis from becoming much enlarged and tender. He had been running an irregular temperature from the time his cystitis began. It varied from 99° to 101°, and was not markedly changed by the development of the epididymitis. The temperature did not return to normal until June 6, a period of about one month.

The first blood count by Dr. Irving, on May 11, was as follows: White blood cells, 8000; differential polynuclears, 76.2 per cent.; lymphocytes, 20.4 per cent.; eosinophiles, 3.2 per cent.; basophiles, 0.1 per cent.

During the first week of the epididymitis he was much more uncomfortable from the acuteness of the inflammation than patients ordinarily are with a gonorrhœal epididymitis. For a short time hot applications were used in place of the cold lead and opium, but were discontinued, as they did not control the pain as well as the ice. On May 23, he had a severe chill, lasting for some time, but not followed by any marked rise of temperature. Pain in the epididymis was severe if the ice was discontinued. Dr. Charles H. Peck was asked to see him, but could find nothing in the kidney or epididymis which would seem to call for operative interference. At this time the blood examined by Dr. Irving showed: White blood cells, 22,000; polynuclears, 87 per cent. There was no sign of any change taking place in the epididymis; it was large and tender, but there were no signs of suppuration. It was decided to use the vaccines which Dr. Jessup had prepared, and 5,000,000 were given in the afternoon of May 24. This was followed shortly by a chill and rise of temperature to 102.8°. Next morning there was another chill, and the temperature rose to 103°. Six days later another injection of 5,000,000 was given, and this time was followed by no reaction. Again on June 5, 10,000,000 were given without causing any disturbance. Later 30,000,000 were given, and only caused a slight reaction.

The exudation into the epididymis was extremely slow in being absorbed, and it was a considerable time before it returned to normal. Soon massage of the prostate and irrigations were begun. The colon bacilli could be found in the fluid obtained after massage for some time afterward, although the urine was perfectly clear, and they seemingly caused no disturbance by their presence.

In this case the epididymitis was due to the colon bacilli, which were found in pure culture in the urine obtained by catheter, and also found in the fluid from the prostate. The examination by Dr. Irving points to a pyelitis as well as the cystitis, which he quite evidently had. In his case there was no question as to the virulency of the inflammation in the epididymis, as I have never seen a gonorrheal epididymitis which was any more severe, and seldom one which was so prolonged as was this one. The use of

the vaccines did not seem to me to be of any special benefit. Possibly if they had been used at an earlier stage the benefit might have been more marked. As an autogenous vaccine was used there could have been no question as to the proper strain.

The persistence of the infection is to be noticed in this case. As the organisms were found in the urine which had not been allowed to remain in the bladder, it would seem a reasonable assumption that they were passing downward from the kidney, and that for some reason, possibly due to the asparagus, they had given rise to the cystitis, with subsequent involvement of the prostate, as shown by the conditions of the gland on examination and the presence of pus and colon bacilli in the massaged fluid. As a result of the prostatitis and posterior urethritis the inflammation extended by continuity and epididymitis resulted. As I review the case it seems fair to conclude that there had not been a sudden invasion of the bacilli, but that in all probability they had been present for some time in the urine without causing any disturbance; but when suitable conditions were present, they were able to set up an inflammation which extended to the structures involved.

THE RATIONAL TREATMENT OF TETANUS, WITH A REPORT OF TWENTY-THREE CASES FROM THE EPISCOPAL HOSPITAL, PHILADELPHIA.[9]

By Astley Paston Cooper Ashhurst, M.D.,

INSTRUCTOR IN SURGERY IN THE UNIVERSITY OF PENNSYLVANIA; ASSOCIATE SURGEON TO THE EPISCOPAL HOSPITAL, ETC.,

AND

Rutherford Lewis John, M.D.,

RESIDENT PHYSICIAN, EPISCOPAL HOSPITAL.

(Concluded from page 819, June, 1913.)

TREATMENT OF TETANUS.

A. The first logical step in the treatment of this disease is the *removal of the source which supplies the toxin*—that is, of the tetanus bacilli. If the point of inoculation is known, it should be attacked directly. The wound should be opened widely, and should be mechanically cleansed of foreign bodies and sloughs. Then it should be swabbed out with a 3 per cent. alcoholic solution of iodine, rinsed with hydrogen peroxide, and filled loosely with gauze soaked in the iodine solution. We believe all caustics should be avoided, as favoring the growth of tetanus bacilli by the formation of sloughs. If the nature of the case demands it for other

[9] Read at a meeting of the Episcopal Hospital Clinical Society, November 18, 1912.

reasons, amputation should be done; then the stump should be left open, and treated as the original wound. Probably in many cases it will be well to follow Porter and Richardson's suggestion to excise the related lymph nodes, particularly if they are palpably enlarged.

B. The next indication is to *head off and neutralize the toxin already formed*. This involves a discussion of the THERAPEUTIC USE OF ANTITOXIN (see this JOURNAL for June, 1913, footnote on p. 814).

Before the introduction of antitoxin as a therapeutic measure the mortality was from 80 to 90 per cent. (Rose, 1897). Collective statistics of cases treated with antitoxin (1897 to 1903), according to Sawamura, showed a death rate of from 28 to 45 per cent. Of course, a collection of isolated case reports is apt to give too low a mortality, owing to the fact that surgeons who have had successful cases will report them, while the larger number do not report their unsuccessful cases. But the fact that no better results were reported from the therapeutic use of antitoxin in tetanus, in view of the remarkable successes from the use of diphtheria antitoxin, was explained on the theory that the antitoxin had not been used early enough. In 1902, Ullrich, according to Sawamura, remedied this apparent defect by collecting 13 cases in which antitoxin had been used early; but only one of these patients recovered; and of 54 collected cases, in which it was used early (subcutaneously), between 1899 and 1905, Busch (Sawamura) found the mortality was 66.5 per cent. In the elaborate statistics collected by Jacobson and Pease the mortality in 191 cases in which antitoxin was used, was 69.6 per cent.; these statistics are especially valuable because they represent series of consecutive cases from various sources. Fricker has published statistics of 40 consecutive cases: in the first 18 (1889 to 1897), no antitoxin was used, and 16 deaths occurred (88.8 per cent.); in the last 22 cases (1897 to 1902), antitoxin was used, and the mortality was only 55.5 per cent. In spite of this decrease in mortality, many physicians have denied that antitoxin has any therapeutic value in acute severe cases.

Huber has reported recently in great detail 69 cases of tetanus treated in Sauerbruch's clinic at Zurich, between 1881 and 1911. Of this whole number, 18 patients recovered and 51 died, a mortality of 74 per cent. Before 1900 antitoxin was not used, and of 31 patients treated without it, 20 died, a mortality of 64.5 per cent. Antitoxin was used in all of the 38 patients treated since 1900, but of these only 7 recovered, while 31 died, a mortality of 81.5 per cent. Huber thinks all of these 7 patients would have recovered even if no antitoxin had been given. But the total amounts used were small (the greatest amount used in any one case was 150 c.c. = 375—750 A. E.), and the mode of administration is open to criticism; this will be discussed later (p. 86).

But here, again, as in the question of its prophylactic use, we must inquire as to the frequency, the quantity, and the site of the injections. In most of the isolated case reports the antitoxin has been injected only once or twice, in small quantities, and subcutaneously.

Exactly how the antitoxin acts upon the toxin is not known. According to Solieri, the idea that its neutralizing action is a direct chemical one, as that of an acid on an alkali, has been abandoned. Dönitz taught that it could not only neutralize the toxin circulating in the blood, but also could loosen toxin already bound in the central nervous system and drive it out of its almost impregnable fortress. According to Metschnikoff, tetanus antitoxin stimulates phagocytosis. From numerous experiments Kraus and Amiradzibi conclude that antitoxin cures by drawing the toxin out of the cells where it is lodged, but does not itself enter into the toxin-containing cells. Recovery under the use of antitoxin they think depends on the possibility of this diffusion; and as such diffusion can take place experimentally through a membrane of collodion and through reeds (Schilfröhren), they think it is not necessary in the human body for antitoxin to be in direct contact with toxin for it to exert its neutralizing influence on the latter.

I. Site of Injection of Antitoxin. The following sites of injection have been advocated: Subcutaneous, intravenous, intraspinal, intraneural, intracerebral, and intramuscular.

1. *Subcutaneous Injections.* This is the method usually employed, but, as already noted, the reports are not encouraging. If administered in this way, the antitoxin is absorbed by the lymphatics, transported to the veins, passes through the lungs, and finally, is distributed through the arterial system to all parts of the body. Only a homeopathic dose ultimately reaches the motor nerves through which the toxin is being carried to the spinal cord, while by far the greater part of the antitoxin is distributed to the viscera, where it can be of no possible use. Administered in this way, overwhelming amounts are required to produce any effect, and it is evidently the height of extravagance so to employ it.

The manufacturers of antitoxin recommend the administration of from 15,000 to 18,000 units (subcutaneously) every three hours; and such an amount (120,000 to 144,000 units) in the course of twenty-four hours is not unreasonable when it is recollected that only a very small fraction of what is administered subcutaneously can be expected to reach the seat of disease, while the rest is a shocking waste of a valuable and very costly remedy. For the amount mentioned (144,000 units) the cost is about $100, even with the discount allowed to hospitals. (One dozen tubes of 1500 units each (18,000 units) cost the Episcopal Hospital, $12.67; eight doses of 18,000 units cost $101.56). Note the history of

Case 23, of our series; here antitoxin was administered subcutaneously in appropriate doses (99,000 units on the first day; 65,000 units on the second day; 60,000 units on the third day)—to a total amount of 224,000 units,[10] and recovery followed. The cost to the Hospital, with discounts deducted, was about $180. The patient was a very poor man, a charity case; but when told of the expense of his treatment, he and his friends collected $10 and donated that sum to the hospital, as a "Widow's Mite."

Compare with this history the case of a severer case of tetanus (Case 11 of our series): On the first day 3000 units were administered intraspinally; on the second day, 750 units were injected into the sciatic nerve, and 750 units deeply into the tissues around the wound. The patient recovered, and the total cost to the hospital was about $3.

The quantities of antitoxin used in each case were logically correct, but only in Case 11 was the mode of administration rational.

2. *Intravenous Injections.* The effects of intravenous injections of antitoxin have been studied experimentally by v. Graff, both as regards prophylactic and therapeutic use:

(*a*) In a first series of experiments he administered the toxin by *intramuscular* injection. (*a*) Antitoxin was administered to 14 rabbits *before* toxin was injected; of these animals only 4 died, and not one had positive signs of tetanus. (*b*) Antitoxin (intravenously) and toxin (intramuscularly) were administered *simultaneously* to 8 rabbits; 4 of these died, but only 1 presented symptoms of tetanus. (*c*) Toxin was administered intramuscularly to 8 rabbits, and *from fifteen to eighteen hours later* antitoxin was injected intravenously; 4 of these rabbits died, 2 with only local tetanus, and 2 with general tetanus.

(*b*) In a second series of experiments he administered the toxin by *intraneural* injection. (*a*) Toxin was injected intraneurally in 6 rabbits; all died of tetanus in from thirty-six hours to five days. (*b*) Antitoxin was administered intravenously to 19 rabbits *from six to ten hours before* a lethal dose of toxin was injected intraneurally. Only 2 of the animals lived and 17 died after intervals of from five to twenty-five days; but in the fatal cases death was attributed to intercurrent gastro-enteritis, no evidence of tetanus having been present. (*c*) Antitoxin was injected intravenously *twenty-four hours before* toxin was injected intraneurally; of 5 rabbits so treated none developed tetanus, 2 lived, and 3 died from

[10] The nearest approach to this total quantity that we can find reported was administered to a patient under the care of Beates and Thomas. The most given on any one day was 97,940 units, the total amount being 213,700 units; but as the patient was a boy of fourteen years (weight 130 pounds), this amount may be relatively greater. Neary administered 280,000 units to his patient, who recovered; but as this quantity was spread out over a period of two weeks (10,000 units every twelve hours), it is not comparable to the doses above mentioned.

no apparent cause in from four to nine days. (*d*) Antitoxin (intravenously) and toxin (intraneurally) were injected *simultaneously;* of 5 rabbits so treated none developed symptoms of tetanus, yet all but 1 died in less than eight days. (*e*) Toxin was injected intraneurally, and *from fourteen to eighteen hours later* antitoxin was injected intravenously; of 3 rabbits so treated, the first developed local tetanus, but recovered, only to die on the ninth day of purulent peritonitis; the second developed local tetanus but recovered; the third developed local tetanus and then general tetanus, and died on the fifth day of general tetanus in spite of intravenous injections of antitoxin repeated every day. (*f*) Toxin was injected intraneurally, and *from fifteen to seventeen hours later* antitoxin was administered intravenously; of 7 rabbits so treated, 4 died as if no antitoxin had been given; in 1 life was prolonged by the treatment until the sixth day, when death from general tetanus occurred; and in 2 recovery ensued, although very severe general tetanus had already developed. These 2 recoveries, v. Graff thinks, are to be explained only on the ground that the antitoxin was able to neutralize toxin already absorbed by the nervous system.

The conclusion of v. Graff is that intravenous administration is the most effective method, but that subdural (intraspinal) administration holds next rank, because he believes in this way antitoxin is rapidly discharged from the cerebrospinal fluid into the blood.[11] Intraneural injections of antitoxin were not employed by v. Graff, but the results of Sawamura's experiments, detailed below (p. 84), as to the relative therapeutic value of intravenous and intraneural administration of antitoxin, seem to demonstrate conclusively the superiority of the intraneural method. From these experiments, to be discussed presently, it is evident that antitoxin administered only by the intravenous method is not very effective; and the same objections as to extravagance apply to this as to the subcutaneous method, only in less degree, because the antitoxin reaches the circulating toxin in shorter time, and can exert its influence sooner on the toxin already absorbed by the peripheral nerves or the spinal cord, if only it is used in sufficient quantities.

That the amount required to produce any therapeutic effect is immense, is proved by the observations of v. Leyden; he neutralized all the toxin in the patient's circulating blood, rendering it harmless to experimental animals; but his patient died of tetanus. And in a patient under the care of Blumenthal and v. Leyden, they succeeded in neutralizing almost entirely the circulating toxin, but much toxin remained in the cerebrospinal fluid. More recent observations tend to show that the amount of toxin circulating in the blood rapidly diminishes after the first few days of the disease, while that in the nervous tissues steadily increases.

[11] v. Graff says that L. Simon has treated 6 consecutive cases of tetanus by intravenous injections of antitoxin, with 4 recoveries.

The following advantages of intravenous administration are pointed out by v. Graff:

(i) The antitoxin gets into the blood as soon as possible.

(ii) This method can be used even when the point of inoculation is doubtful, or unknown, or inaccessible. He recommends it especially for cases of puerperal origin.

(iii) It is easier than intraspinal or intraneural injection, is less painful, and can be repeated.

As regards the first advantage, it may be replied that it is more important to have the antitoxin in the nerves and spinal cord than in the blood. Intravenous administration is not the only available method for cases mentioned in the second paragraph, since intraspinal (subdural) injections may be made; and these, as well as intraneural injections can be repeated as often as is desirable. However, they are more painful to the patient, sometimes requiring a general anesthetic, and intraneural injections require more accurate anatomical knowledge and surgical skill.

3. *Intraspinal (Subdural) Injections.* These were originally suggested by Jacob; but he came to the conclusion, after some experimental work, that they were useless, because he thought all the antitoxin so administered rapidly escaped into the general circulation. Sicard, however (quoted by Hofmann), obtained better results in his experiments with dogs. The method was first used successfully in 1899 by v. Leyden, who thought this the best method of all, emphasizing the fact that the antitoxin thus came into intimate relation with blood- and lymph-vessels of the cord, and so was conveyed more quickly to the medullary cells. Whether or not antitoxin injected into the subdural space of the cord acts directly upon the cord itself, or upon the nerve roots, does not appear to have been determined. Certainly it has not been disproved, and if reasoning by analogy with tetanus toxin be allowed, it is altogether probable that antitoxin injected intraspinally is in large part absorbed into the nerve roots and the cord itself, especially if the pia or the nerve tissue is punctured. Mere withdrawal of cerebrospinal fluid by lumbar puncture has been suggested as a therapeutic measure, and it is possible that the rapid decrease in severity of the symptoms often seen may be produced in this way. It is not reasonable to expect the specific action of the antitoxin to be manifested for several hours.

Though, experimentally, treatment of tetanus by intraspinal injections of antitoxin has not been very encouraging, much more satisfactory results have been obtained in actual practice.

Hofmann reported from v. Hacker's clinic, at Graz, a series of 30 consecutive cases of tetanus. From theoretical considerations they were opposed to the intraspinal administration of antitoxin, and in the first 13 cases only subcutaneous injections were used; of these patients 7 died, a mortality of 53.8 per cent. In 3 of these

cases, 2 of them fatal, carbolic acid was also employed subcutaneously. In a fourteenth case, which also died, both subcutaneous and intraneural injections were given. Of the cases treated by subcutaneous injections only, 5 came under treatment within thirty hours of the onset of symptoms; and of these patients (mostly acute cases), 4 died, a mortality of 80 per cent.; 8 cases came under treatment more than thirty hours after the onset of symptoms, and of these only 3 patients died, a mortality of 37.5 per cent. When the fifteenth patient came under treatment, intraspinal injections of antitoxin were tried, because other treatment did not control the disease, which was of the acute type, and very severe. This patient recovered. Then in the succeeding 15 patients antitoxin was always given intraspinally; and *of the entire series of 16 patients treated by antitoxin subdurally, only 2 died*, a mortality of 12.5 per cent.; 4 of these were very acute cases, yet all recovered. In 2 cases of this latter series antitoxin was injected intraneurally as well as intraspinally.

Rogers, in a series of 7 acute cases, employed intraspinal as well as intraneural injections, and in administering the intraspinal injections he endeavored to inject at least some of the antitoxin directly into the spinal cord (intramedullary injection), so that it might reach the nerve centres as quickly as possible. Of his 7 patients, only 3 died. Of 5 patients under the care of Luckett, all the 4 who were treated by intraspinal injections recovered, while the fifth patient, not so treated, died.

In our own series of cases intrapsinal injections of antitoxin were employed only in 7 patients, of whom 4 died, a mortality of 57 per cent.; but in 1 fatal case the injection was made only three hours before death, as a last resort (Case 2); in another, death was caused by a subsequent overdose of magnesium sulphate, injected into the subdural space (Case 19); and the 2 other deaths occurred in extremely acute cases (Cases 12, 13). In all the successful cases improvement was rapid (Cases 8, 10, 11).

4. *Intraneural Injections.* This method was first employed clinically by Küster in 1902. Jacobson and Pease suggested that the pressure, rather than the specific action of the antitoxin, blocked further absorption of the toxins; but an experiment in which Sawamura employed salt solution instead of antitoxin for intraneural injection, seems to disprove this supposition.

As it is a well ascertained fact that most, possibly all, of the toxin reaches the spinal cord only by travelling up its nerves, it is theoretically logical to inject the antitoxin into the nerves, in order that, like the toxin, it may reach the spinal cord and rout the enemy by the easiest road. That it will do this, when injected intraneurally, admits of no doubt in view of the overwhelming experimental evidence on the point. Just how the antitoxin acts, and by which intraneural route (axis cylinders or endo- and peri-

neurium) it reaches the cord, has not been determined. Now, though most of the toxin ascends the nerves leading from the wounded part, other lesser amounts of the toxin are simultaneously invading the cord through all the motor nerves of the body; and it is manifestly impracticable to expose and inject antitoxin into all of these nerves. Moreover, when the site of inoculation is doubtful, or unknown, uncertainty must exist as to which nerves should have injections of antitoxin. The only methods we possess for reaching all the nerves at once are (1) intravenous injections, and (2) intraspinal injections. In no case, therefore, should the surgeon depend upon intraneural injections alone, and in no case should he omit either intraspinal injections or injections into the motor nerves leading from the site of inoculation. Especially if the point of inoculation is in a muscular part, or in the upper extremity, will the main bulk of the toxin reach the cord by the motor nerves of the inoculated part; and in such cases particularly are intraneural injections requisite.

Sawamura conducted experiments to determine the relative therapeutic value of antitoxin administered *intravenously* and *intraneurally*. (1) In a first series of 4 rabbits he administered antitoxin *intravenously from eighteen to nineteen hours after* toxin had been injected intramuscularly; all 4 rabbits died of tetanus. (2) In a second series of 6 rabbits he administered antitoxin either *intraneurally alone* or *intraneurally as well as intravenously, from eighteen to twenty-four hours after toxin* had been injected intramuscularly; and although the *total amount of antitoxin* was no greater or even less than that which had been employed in his first series, yet only 2 of the rabbits died, and these deaths did not occur until the twelfth and the sixteenth days, in cases where the total amount of antitoxin was small, where it had been injected late, and where it had been given only intraneurally, and not intravenously as well. (3) In a third series of 3 rabbits he administered antitoxin *intravenously* forty hours after the intramuscular injection of toxin, and seventeen hours after the appearance of the first symptoms of tetanus; he administered antitoxin in large amounts *intravenously*, *intramuscularly*, or *subcutaneously*, or *by all three methods*, and *on several subsequent days;* yet *all these rabbits died of tetanus in from nine to fourteen days.* (4) In a fourth series of 4 rabbits he administered *a much less amount of antitoxin intraneurally*, after the same interval since the intramuscular injection of the toxin as in the third series; *yet not* 1 *of these* 4 *rabbits died.* In another rabbit, similarly prepared, for the sake of a control experiment, he injected salt solution into the nerve instead of antitoxin; but this rabbit died of tetanus on the ninth day.

Sawamura says (p. 85): "To increase the value of intraneural injections of antitoxin, one must strive not only to neutralize the toxin in the peripheral nerves or that which is later to reach them,

but also to bring the action of the antitoxin to bear on the toxin already in the spinal cord; and, therefore, the injection should be made into the nerve as near the cord as possible; and centrally from this point as large an amount of antitoxin as is possible must be injected." He thinks that if in his earlier experiments (the second series quoted above) he had injected the antitoxin into the central instead of into the peripheral part of the nerves, he would have had still better results.

There are few clinical reports of cases treated by intraneural injections of antitoxin; most of them are isolated cases, and hence are of little value. Küster's patient, the first so treated, recovered. Rogers has employed this plan in 7 cases, with 4 recoveries and 3 deaths, a mortality of 43 per cent. (In all these cases intraspinal injections were employed also.) Sawamura, in 1909, collected 12 isolated cases of tetanus, treated by intraneural injections of antitoxin, with only 4 deaths, a mortality of 33 per cent. In our own series of cases it was employed only twice (Cases 11 and 13), only the first patient recovering.

In the patients who have recovered after this treatment, no disability appears to have been caused except in Küster's case, where some neuritis and muscular atrophy and contractures occurred; and these may have been due to the effects of the original injury and not attributable to the treatment employed. Our own patient suffered no inconvenience from the injection made into his sciatic nerve.

5. *Intracerebral Injections.* These were originated by Roux and Borrel, who found the method valuable in experimental work: of 45 guinea-pigs thus treated, 35 recovered; whereas of 17 treated by subcutaneous injections of antitoxin only 2 recovered. But when applied to mankind, the results have been absolutely bad—not only has the mortality not been reduced, but lasting cerebral lesions have been produced in some of the patients who recovered. In 88 cases collected by v. Graff, in which intracerebral injections were used, there were 71 deaths, a mortality of over 80 per cent. Sawamura quotes the following statistics for intracerebral injections: Lambert, 52 cases, 63.43 per cent. mortality; Lereboullet, 26 cases, 67.5 per cent. mortality; Steuer, 55 cases, 67.3 per cent. mortality.

6. *Intramuscular injections* have not been employed in any large series of cases. Antitoxin injected around the wound, whether as a prophylactic or as a therapeutic agent, should be given into the muscular tissues whenever possible, to afford it a readier access to the motor nerves. As a therapeutic method intramuscular injection is better than the subcutaneous, but is perhaps inferior to the intravenous, and certainly is inferior to the intraspinal and intraneural methods.

II. Frequency of Injection of Antitoxin. The series of cases reported by Huber has already been referred to. He thinks the antitoxin employed was of no value, and when we come to study his case reports it is quite evident that in most cases this is true. But it is true because antitoxin was not given often enough to be of any service, as well as because the total quantities were absurdly small, in view of the fact that most of it was injected subcutaneously. In 17 cases one injection alone was given, and 1 patient recovered. In 16 cases two injections were given, and 4 patients recovered. In 2 cases three injections were given, and none recovered. In 3 cases four injections were given, and 2 patients recovered. Nor is it true that multiple injections were not given because the patients did not survive long enough. Even a patient who dies in twenty-four hours after coming under treatment lives long enough to receive at least eight subcutaneous injections. When it is administered intravenously larger amounts can be given at one time, and one or at the most two injections in twenty-four hours should suffice. Intraspinal injections seldom are requisite more than once daily, and often only every third day. Intraneural injections can be repeated daily if required; this was done on three consecutive days in Case 13 of our series, but unfortunately without a successful result.

III. Quantity of Antitoxin Injected. No matter what the method of injection, *the most important thing is to get the maximum quantity of antitoxin indicated into the patient's body as soon as possible*. Delay even of a few hours may determine a fatal result; 25,000 units given within the first three hours almost certainly are of more use than 50,000 units given after six hours, or given in divided doses. If one determines to use antitoxin at all, he should, we believe, make it a rule to administer it as early as possible, and *to administer the total quantity indicated as nearly as may be all at one time*. There can be scarcely any doubt that in most of the reported cases, as for example, in Huber's series, the amount of antitoxin administered has been utterly inadequate.

As already pointed out, if the injections are given *subcutaneously*, immense quantities are indicated. For an adult, with the usual acute type of case, at least 100,000 units are required in the first twenty-four hours; though a less amount may be sufficient for a child or for a comparatively mild case, one cannot be sure of the fact, and it is better to give too much than not enough. Administered *intravenously*, a less amount is sufficient; how much it is difficult to say. Probably 15,000 to 25,000 units should be administered at first, and if no effect is apparent, or if the good effect wears off, a similar amount should be given after the lapse of eighteen to twenty-four hours. If given *intraspinally*, from 3000 to 10,000 units should be given, according to the severity of the case; this need not, as a rule, be repeated in less than eighteen

to twenty-four hours. Even when administered intraspinally a certain interval must elapse before the effect of the antitoxin can be apparent. *Intraneural* injections should be made in as great amounts as the nerves will absorb. We have injected 1500 units into the sciatic nerve, all at once, on several occasions, and 750 units into each of the anterior crural and obturator nerves. If the injections are slowly made practically all of this quantity can be introduced among the nerve fibers.

Carbolic Acid Injections in the Treatment of Tetanus. This method was first brought to the attention of the profession in 1893 by Bacelli, who had employed it since 1888. The well-known anesthetic properties of phenol indicate that it has an affinity for nervous tissue, and it had been used successfully by Bacelli and others in cases of neuritis before he adopted it in cases of tetanus. According to Imperiali, carbolic acid is both antibacterial and antitoxic to the Bacillus tetani, and acts, moreover, as a nervous sedative. He has collected 190 cases treated by Bacelli's method, with 157 recoveries and 33 deaths, a mortality of only 17.3 per cent. He classified the cases thus:

		Mortality.
Severe cases,	94, with 2 deaths and 92 recoveries.....	2.1 per cent.
Severest cases,	39, with 17 deaths and 22 recoveries.....	43.5 per cent.
Fulminating cases,	15, with 14 deaths and 1 recovery	93.3 per cent.

These statistics are open to the usual objections applicable to collected cases; and it is strange that among so large a number of case reports no cases appear which may be classed as mild or chronic in type. It may be that the Italians, recognizing that everything is comparative, class as severe those cases which we consider mild, on the theory that even a mild attack of tetanus is a severe disease. Imperiali, however, quotes Meoni as having observed 6 cases of tetanus in the past four years, all treated by carbolic injections, and with only one death. Surgeons in other countries, however, have not reported as successful results. Pearce Kintzing, almost alone in this country, reports favorable results; he treated 7 patients by carbolic injections, and all recovered; in 3 of these cases the onset of the disease was acute, in 3 the onset is not described, and in 1 the onset was very gradual. In less than half of his cases could the disease be considered very severe in type.

Bacelli's plan is to administer 1 c.c. of a 1 per cent. solution every few hours, preferably into the muscles along the spine, until 80 or 100 centigrams are given in twenty-four hours.[12] In none of the cases mentioned by Imperiali did the total amount admin-

[12] Most preparations of antitoxin in this country have added, as preservative, 0.5 per cent. of trikresol (Hitchens). Some of the foreign sera are said to contain 0.5 per cent. of phenol. Any therapeutic effect this may have must be beneficial in cases of tetanus.

istered in twenty-four hours exceed 50 centigrams. Kintzing made a 10 per cent. solution by dissolving the deliquesced crystals of phenol in sterile water; the full adult dose was 10 drops of this 10 per cent. solution (equivalent to 1 grain of pure phenol), which was diluted in 30 or 40 minims of sterile water, just before being injected. Most physicians have preferred to use a weaker solution (0.5 per cent.), and to make the injection every one or two hours, carefully watching for constitutional symptoms of carbolic acid poisoning. The tolerance of tetanus patients for carbolic acid is amazing.

Phenol injections were employed in only one patient in our series (Case 20), in conjunction with small amounts of antitoxin subcutaneously. It was a mild, chronic type of case, of puerperal origin, and though the patient survived the disappearance of all tetanic symptoms for a period of nine days, she eventually died from puerperal sepsis.

Camus has made a series of experiments to determine the comparative value of carbolic acid, magnesium sulphate, and antitoxin in the treatment of tetanus. Dogs were used, and the animals in each series received exactly the same amount of the same toxin, at the same time. These investigations show that magnesium sulphate (intraspinally) and carbolic acid (subcutaneously) have no influence on the evolution of tetanus, no matter in what amounts or at what stage of the disease they were administered. Magnesium sulphate has no other action than as a spinal depressant; and while carbolic acid possibly may have some antibacterial action, it has no effect on the fixed toxin nor on the toxin in course of fixation. Antitoxin, alone, injected simultaneously into the cerebrospinal fluid by lumbar puncture, intravenously, and subcutaneously, *gave very much better results.*

Cholesterin Injections in the Treatment of Tetanus. Experiments having shown that it is the cholesterin of the central nervous system for which tetanus toxin has special affinity, it occurred to Almagia and Mendes that hypodermic injections of this substance might neutralize the toxin. In 2 patients they used successfully injections of from 15 to 30 centigrams of cholesterin in 10 c.c. of distilled water; Pribram, however, reports the use of cholesterin in 3 patients, all of whom died. Among other substances which have been found experimentally to be capable of neutralizing tetanus toxin is urea (Sewaki).

C. The third indication in the treatment of tetanus is *to depress the functions of the spinal cord.* So far we have considered only methods to check the supply of toxin and to neutralize the toxin already formed. Now we come to an equally important factor, because even if the supply of toxin can be stopped promptly, and even if the toxin not yet firmly bound to the nerve tissues can be completely neutralized, there is in almost every case a

large amount of toxin which has become impregnably entrenched in the central nervous system, particularly in the spinal cord, and *none of the methods of treatment hitherto discussed has any influence over it*. Until its action is exhausted it continues to stimulate the motor, and to a less degree, the sensory tracts of the spinal cord, and kills the patient by exhaustion. The only way to remedy this state of affairs, so far as we know, is to depress the functions of the spinal cord. We have at our disposal a number of drugs whose main therapeutic action is to render the spinal cord less susceptible to stimulus. Administration of one or more of these remedies forms an integral part of any rational plan for the treatment of tetanus. The drugs most often employed are chloral, chloretone, and similar products; the bromides; physostigma, hyoscine, morphine, and magnesium sulphate.

The usual doses of these drugs, recommended for ordinary occasions, do not apply to the treatment of tetanus. The object is to give enough of the drug to produce therapeutic action, and until this has been obtained the dose may be steadily increased. But there is one caution always to be kept in mind: this is, that the gastro-intestinal tract of a patient with tetanus may not absorb as readily as might be expected, and there is danger that drugs will accumulate in the bowels and be suddenly absorbed in overwhelming doses when a turn for the better occurs. This may be theoretical reasoning, but we are sure we have seen more deaths from tetanus with the patient completely relaxed than in convulsions. In our series of 23 cases (13 deaths) the condition at death is noted in all but 4 (Cases 1, 7, 17, 21); only 3 (Cases 4, 16, 18) died in spasm or convulsion, and 6 (Cases 2, 5, 12, 13, 19, 22) died in complete relaxation; and in some of these cases the condition was due to overaction of the spinal depressants employed.

We believe most reliance can be placed on the use of *chloral* and the *bromides*. Hutchings employed *chloretone* in 6 cases with only 1 death, and he warmly advocates this drug. It is administered by mouth or rectum in doses of from 30 to 60 grains, dissolved in whisky or in hot olive oil. In our series it was employed three times: Case 12 died after being comatose for twenty-four hours; Case 13 died after being comatose for three hours; Case 20 died from puerperal sepsis. In both Cases 12 and 13 the administration of chloretone was stopped as soon as the patients began to show signs of coma, but without avail.

Morphine as the only depressant was used in only one case in this series (Case 21), the patient dying. We believe it is less effective as a spinal depressant than chloral and the bromides, and that it never should be employed to the exclusion of these drugs. In combination with other spinal depressants it was systematically employed only in Cases 1, 5, 22, and 23, three patients dying.

Hyoscine was used in Case 15, the patient recovering.

Chloral and bromides were employed in practically all cases except Case 21, in which morphine alone was used.

Magnesium Sulphate. In 1906, Blake adopted intraspinal (subdural) injections of magnesium sulphate in cases of tetanus. The treatment is based on the anesthetic effect of intraspinal injections of this drug, as determined by Meltzer in 1905. A 25 per cent. watery solution of the chemically pure drug is employed, and 1 c.c. of the solution is used for every twenty-five pounds of body weight. In heavy adults, however, this dosage might prove excessive, as it might also in some women and young children. Fox, in 1910, collected 24 cases which had been treated by intraspinal injections of magnesium sulphate. Among these patients there were 11 deaths, a mortality of 45.8 per cent. Such isolated case reports have little value, as they give no true idea of the value of a remedy. Only when some large consecutive series of cases has been treated with magnesium sulphate can we determine its proper place in the treatment of tetanus. According to Fox's table, J. N. Henry is the physician who has had most experience with this method of treatment. He reported 4 cases, with 3 deaths, 1 of which may have been caused by magnesium-sulphate poisoning. In our own series of cases intraspinal injections of magnesium sulphate were employed in 3 instances (Cases 17, 19, 22), and all the patients died, 1 (Case 19) undoubtedly of an overdose. Other cases have been reported in which death was attributed to the remedy rather than the disease (Henry, Hessert), or in which a fatal termination was averted solely by resort to active stimulation and artificial respiration (Soutter).

Magnesium sulphate has also been used subcutaneously. Miller quotes 3 mild cases with recovery, and Paterson reports 1 successful case.

The experiments of Camus, concerning the therapeutic value of magnesium sulphate, have already been mentioned.

D. *The patient*, as well as the disease, must be treated; and we come finally to say a few words about feeding, nursing, etc. When first seen it is well to administer a purge; when the disease is once fully established, it may be very difficult to secure an evacuation of the bowels. Retention of urine must be watched for and relieved by the catheter. Isolation is desirable rather for the sake of protecting the patient from noise than for the purpose of preventing contamination of other patients, which is very rare.[13] Slamming doors, loud and especially sudden talking, and in fact noises of all kinds should be prevented. The patient's ears may be stopped with cotton, and the floor heavily carpeted. A bed that squeaks

[13] Reynier says that in 1902 his assistant carried spores on his ungloved hands from one hospital, where he had amputated for tetanus, to another hospital where he infected 3 patients who died of tetanus after operations by this assistant. Now that gloves are habitually worn, such an occurrence could hardly take place.

during the convulsions is a great annoyance. Window frames should be kept from rattling. Nursing must be constant and painstaking. Food must be administered at all hazards, by a nasal tube if necessary. Saline solution by the bowel, as in cases of peritonitis, tends to overcome the dehydration of the tissues produced by excessive muscular activity. Drugs may be administered in the rectal infusion, hypodermically, or by mouth.

SUMMARY. From the foregoing discussion it is evident that the rational treatment of tetanus comprises the four indications enumerated at p. 812. The *care of the wound*, both as a prophylactic and as a curative measure, is most important. The *neutralization of the toxin*, by the rational use of antitoxin, is indispensable; and we think we have demonstrated the inadequacy of the dosage usually employed for subcutaneous administration, and the necessity of intraneural, intraspinal, and probably also of intravenous injections. The excellent results reported in some quarters from the use of *carbolic-acid injections* should be remembered; it is a remedy much more readily obtainable than antitoxin. The third indication, *to depress the functions of the spinal cord*, must not be met to the exclusion of the foregoing. Those who are enthusiastic in the use of intraspinal injections of magnesium sulphate seem to forget that unless they also employ antitoxin in a rational manner they are doing nothing to aid the body tissues to withstand the onslaught of the disease. Finally, the *care of the patient*, nursing and feeding, is the most practical part of the treatment, and one without which all the other parts may fail of their effect.

When we encounter another case of tetanus, we hope to be able to apply rational treatment in the following manner:

The patient will be placed in quiet, with competent nursing facilities. As soon as possible after coming under observation, whether this be in the small hours of the night or at bright noon tide, the motor nerves leading from the wounded part will be exposed, as near to the cord as practicable, and as much antitoxin as each will contain will be injected toward the spinal cord.[14] An intraspinal injection of at least 3000 units will then be made according to the usual technique for spinal anesthesia. If it is possible to prick the cord with the needle, so much the better. Next the wound of entrance of the infection will be widely opened, all foreign bodies, sloughs, etc., will be removed by forceps, scissors, or scalpel; the wound will be irrigated with hot peroxide of hydrogen, swabbed

[14] For wounds of the *sole of the foot*, it is sufficient to inject the sciatic nerve; for those of other parts of the *lower extremity*, not alone the sciatic but the anterior crural and obturator nerves as well should be injected. For wounds of any part of the *upper extremity*, the brachial plexus should be exposed above the clavicle, and an injection should be made into each of its cords. These operations should be done under general anesthesia, for which we prefer chloroform. A strong linen ligature is to be looped loosely around each of the nerves exposed; the ends of these ligatures are to be left long, and used to identify the nerves and draw them up into accessible positions for the purpose of subsequent injections of antitoxin should these prove necessary.

out with 3 per cent. alcoholic solution of iodine, and loosely filled with gauze soaked in the same solution, and injection of antitoxin will be made (1500 to 3000 units) deeply into the muscular tissues around the wound. Continuous proctoclysis, as used in cases of peritonitis, will be given; and by mouth or in the rectal fluid will be administered effective doses of chloral and bromides, at appropriate intervals. Feeding will be enforced, by the nasal tube passed under chloroform anesthesia, if necessary. During the course of the first day a moderate amount of antitoxin will be administered intravenously; probably 10,000 units will suffice.

The intraneural and intraspinal injections of antitoxin will be repeated daily, under chloroform anesthesia, until marked decrease in spasticity occurs. Every twelve hours, or less often, a moderate amount of antitoxin will be injected intravenously, or even subcutaneously, so as to neutralize the circulating toxins; but the main reliance will be placed on intraneural and intraspinal injections. The administration of spinal depressants will be continued so long as they are indicated; a comatose state or muscular relaxation naturally are contraindications. The wound will be dressed daily, as above described, until a healthy granulating surface is obtained.

With such treatment, commenced within twelve hours of the first appearance of symptoms of tetanus, we believe the mortality of the disease should not be over 20 per cent. Of the 11 patients under our own care, 7 have recovered and only 4 died, a mortality of 36.36 per cent. One of these deaths was caused by an overdose of magnesium sulphate; this patient did not come under observation until the fourth day of the disease, and none of the other fatal cases came under our care until more than twenty-four hours after the onset of indubitable symptoms of tetanus.[15]

[15] Since the above was written, one of us (Ashhurst) has seen in consultation with Dr. George W. Norris, in his ward at the Pennsylvania Hospital, another case of tetanus:

Samuel W., negro, aged twenty-seven years, crushed his right index finger shortly before December 1, 1912. The skin was broken, but he bandaged the finger himself and never had any medical treatment for it. About December 8 or 9 he began to complain of stiffness and soreness in jaws, but never had any difficulty in swallowing. On December 11 he complained of a "ball of wind" in the epigastrium, which caused oppression and dyspnea (tetanus of diaphragm?). Dyspnea became more urgent on December 13, and on December 14 he went to bed. About this time he noticed that his legs were getting stiff (descending tetanus), and could be flexed only with great difficulty; no complaint of pain. Since December 15 he has had frequent spasms (ten to twelve daily) of his abdominal and thoracic muscles. These caused no pain, but made it more difficult to breathe. His legs were not weak, and would support him if he was helped up into a standing position; but they were perfectly helpless from rigidity.

Admitted to the Pennsylvania Hospital December 17, evening. Receiving ward diagnosis: *Transverse myelitis*. Transferred to medical ward and seen by Dr. Norris on the morning of December 18, when a diagnosis of *tetanus* was made.

First seen by Dr. Ashhurst at 6.45 P.M. on December 18. No antitetanic treatment had been instituted. During the day the patient had had five general convulsions, and had a sixth convulsion while under observation. When examined after this convulsion there was *no trismus;* the head was retracted and the lumbar spine arched; the lower extremities were in full extension and very rigid. The patient lay on his right side.

December 18. At 7.30 P.M., under chloroform anesthesia, lumbar puncture was done: there was a very free flow of spinal fluid, which was under great tension (5 c.c. were sent to the labora-

The following table gives the general mortality in some recent series of consecutive cases of tetanus:

(Consecutive) Case Reports.

	Cases.	Recovered.	Died.	Mortality.
Bockenheimer (1908)	20	3	17	85.0 per cent.
Busch (1907)	30	9	21	70.0 per cent.
Episcopal Hospital (1905 to 1912)	23	10	13	56.5 per cent.
Fricker (1897 to 1902)	22	9	13	55.5 per cent.
Hessert (1909)	15	5	10	66.6 per cent.
Hofmann (1907)	30	20	10	33.3 per cent.
Huber (1912)	38	7	31	81.5 per cent.
Hutchings (1909)	6	4	2	33.3 per cent.
Jacobson and Pease (1906)	191	58	133	69.6 per cent.
Kintzing (1911)	7	7	0	
Magula (1911)	33	11	21	66.7 per cent.
Simon (1911)	6	4	2	33.3 per cent.
Suter (1905)	14	2	12	85.7 per cent.

tory for examination as to presence of tetanus toxin, but through some misunderstanding this examination was not made; a culture of the fluid remained sterile); 8000 units of antitoxin (15 c.c.) were injected into the subdural space of the cord; this was all the antitoxin available at that time. The patient was given chloral, grains xv, and potassium bromide, grains xxx, every three hours by mouth.

At midnight the patient was seen again, and 15,000 units of antitoxin were given intravenously in a pint and a half of saline solution. No more convulsions had occurred.

December 19. The next morning the patient was better. There had been no more convulsions, but many opisthotonic spasms. At 7.30 A.M. he was given a sponge bath, and after this he had two general convulsions. There was more trismus than at any time, and the tongue could just be protruded between the teeth. During the forenoon chloroform was again administered, and Dr. Norris attempted to give antitoxin intraspinally, but was unable to introduce the needle. Therefore 13,500 units were given intravenously in six ounces of saline solution. This made a total of 36,500 units administered in an effective manner within sixteen hours of the time the patient came under surgical observation. No intraneural injections were given, as no facilities existed for a surgical operation.

The patient was seen again at 7 P.M. There had been no convulsions all day. His muscles were quite relaxed. The chloral was reduced.

December 20. Has had no more convulsions; only a few, and not severe, spasms. Has taken food well. Chloral stopped.

December 21. Perfectly relaxed. *Cured of tetanus.* Clear in head and converses normally, but has pneumonia at right base (probably from inspiration of food) and is quite weak.

December 22. Died at night from pneumonia; temperature, 101° F.; pulse, 148; respirations, 40.

Cases of Tetanus at Episcopal Hospital 1905 to 1912.

Number.	Date of admission.	Age and sex.	Site of wound.	Date and nature of injury.	Incubation (time from injury to symptoms.)	Time from symptoms to institution of efficient treatment.	Type of case.	Summary of treatment.		Result.	Service of	Attending.
								Original injury.	Tetanus.			
1	Oct. 9, '05	23, M.	Left arm	Sept. 18, '05. Vaccination.	Unknown. Less than 19 days	3 days	Severe. Convulsions early.	Neglected.	Sedatives. Antitoxin, subcutaneously (60 c.c. = 18,000 units?) in 1 day.	Died, Oct. 10, '05	Sinkler	Sinkler and Sweeney.
2	June 4, '06	Boy	Left forearm	May 26, '06. Laceration from kick of horse.	9 days	12 hours	Severe. Convulsions early.	Ordinary antiseptic dressing.	Sedatives. Antitoxin, subcutaneously, and (3 hours before death) intraspinally (1500 units). Total amount 45,450 units in 2 days.	Died, June 6, '06	Neilson	Owen.
3	Aug. 6, '06	48, M.	Right hand	Aug. 6, '06. Compound fracture of wrist-joint. (Machinery crush.)	9 days	12 hours	Mild. No convulsions.	Ordinary antiseptic dressing.	Sedatives. Antitoxin, subcutaneously, 108,000 units in 2 days.	Recovered, Aug. 23, '06	Deaver	Weber and Aufhammer.
4	Mar. 5, '08	44, M.	Right toe	Jan. 9, '08. Cut by axe in felling a tree.	8 weeks	3 days	Severe. Convulsions early.	None for 5 weeks. Ordinary antiseptic dressing for 3 weeks.	Toe amputated. Antitoxin subcutaneously, 12,000 units in 1 day.	Died, Mar. 6, '08	Davis	Davis and Brown.
5	May 31, '08	26, F.	Left leg	May 31, '08. Compound fracture. Thrown into river by explosion.	Over 2 weeks	5 days, June 23, '08	Slow in onset, Convulsions late.	Ordinary antiseptic dressing.	Sedatives. Antitoxin, subcutaneously, 18,000 units in 5 days.	Died, June 30, '08. In coma 3 days before death	Neilson	Neilson and Price.

6	Aug. 12, '08	13, M.	Behind ear	Aug. 6, '08. Punctured wound by rusty knife.	4 days	2 days	Mild. No convulsions.	Puncture sutured; no drain.	Antitoxin subcutaneously, 3000 units in 2 days.	Recovered, Aug. 23, '08	Deaver	Deaver and Corson.
7	Aug. 28, '08	35, M.	Foot	Aug. 24, '08. Punctured wound by rusty nail.	5 days	1 hour	Severe. No convulsions.	No treatment for 3 days, then opened and drained.	Sedatives. Antitoxin, subcutaneously, 5000 units in 2 days.	Died, Aug. 30, '08	Deaver	Deaver and Corson.
8	Sept. 28, '08	34, M.	Abdomen and thigh	Sept. 24, '08. Brush burns, hematoma. Thrown from wagon and dragged.	14 days	1 hour	Slow in onset, but severe convulsions.	Ordinary antiseptic.	Sedatives. Antitoxin, subcutaneously, (129,000 units) and intraspinally (3000 units). Total antitoxin 132,000 units in 10 days.	Recovered Oct. 31, '08	Frazier	Ashhurst and Corson.
9	Sept. 23, '08	55, M.	Cranium	Sept. 23, '08. Gunshot fracture of temporal region and rupture of eye-ball.	4 weeks	12 hours	Mild. Slow in onset. No convulsions.	Aseptic. No operation.	Sedatives. Antitoxin, subcutaneously, (31,500 units in 8 days).	Recovered. Eyeball enucleated, Nov. 17, '08	Frazier	Ashhurst and Aufhammer.
10	Nov. 12, '08	49, M.	Foot	Nov. 4,' 08. Punctured wound by rusty nail.	8 days	24 hours	Medium severity. No convulsions.	Home treatment, by ham fat, etc.	Sedatives. Antitoxin, intraspinally (3000 units), and later subcutaneously (27,000 units). Total amount, 30,000 units in 6 days.	Recovered, Nov. 18, '08	Frazier	Ashhurst and Gracey.
11	Dec. 19, '08	19, M.	Foot	Nov. 30, '08. Punctured wound by splinters from mill floor.	18 days	24 hours	Severe. Convulsion before admission.	Wound dressed twice but patient never returned for further dressings.	Three splinters removed from wound. Sedatives. Antitoxin intraspinally, intraneurally, and into muscles around wound. Total in 2 days 4500 units.	Recovered, Dec. 31, '08	Frazier	Ashhurst and Gracey.
12	Oct. 16, '09	63, M.	Fingers	Sept. 28, '09. Lacerations by machinery.	18 days	24 hours	Severe. No convulsions.	Ordinary antiseptic dressings.	Fingers amputated. Sedatives (chloretone). Antitoxin intraspinally, 1500 units.	Died, Oct. 19, '09 After being comatose for 24 hours	Frazier	Ashhurst and Hopper.

Number.	Date of admission.	Age and sex.	Site of wound.	Date and nature of injury.	Incubation (time from injury to symptoms).	Time from symptoms to institution of efficient treatment.	Type of cases.	Summary of treatment.		Result.	Service of	Attending.
								Original injury.	Tetanus.			
13	Nov. 20, '09	11, F.	Knee	Nov. 11, '09. Abrasion from fall.	9 days	40 hours	Slow in onset. Severe. Convulsions late.	Bread-poultice at home.	Sedatives (chloretone). Antitoxin, intraneurally, 3 times (total 9000 units), intraspinally (1500 units), subcutaneously (3500 units). Total antitoxin 13,500 units in 3 days.	Died, Nov. 22, '09 After being comatose for 3 hours	Frazier	Ashhurst and Hopper.
14	Feb. 4, '10	45, F.	Toes	Jan. 23, '10. Laceration. Fall down stairs in bare feet.	12 days	Few hours	Mild. Slow onset. No convulsions.	Neglected for 10 days. Then iodine.	Sedatives. Antitoxin, subcutaneously, 6000 units in 3 days.	Recovered, Feb. 19, '10	Morris	Ashhurst and Siner.
15	Feb. 21, '10	23, F.	Scalp	Jan. 26, '10. Lacerated wound. Hit by brick.	19 days	1 week	Slow in onset. Severe. No convulsions.	Sutured, no drain.	Abscess opened. Sedatives. Antitoxin, intraspinally, 3000 units; subcutaneously, 21,500 units. Total antitoxin 24,500 units in 5 days.	Recovered, Mar. 8, '10	Neilson	Ashhurst and Siner.
16	July 17, '10	8, M.	Thumb	July 5, '10. Cut by hatchet.	10 days	2 days	Slow in onset. Severe. Convulsions.	Sutured, no drain.	Sedatives. Antitoxin, subcutaneously, 103,000 units in 3 days.	Died, July 20, '10 in spastic state	Deaver	Griffith
17	Oct. 14, '10	7, M.	Arm	Sept. 24, '10. Vaccination.	Unknown Less than 3 weeks	12 hours	Mild. Slow in onset. No convulsions.	Neglected.	Sedatives, and magn. sulph. intraspinally. Antitoxin subcutaneously, 60,000 units in 2 days.	Died, Oct. 16, '10	Frazier	Frazier and Henneberger.

						3 weeks		No convulsions.		Antitoxin subcutaneously, 60,000 units in 2 days.			Hennebergeг.
18	Oct. 3, '10	6, F.	Arm	Sept. 7, '10. Vaccination.	Unknown Less than 24 days	3 days	Slow onset. Very severe. 2 convulsions every hour.	Treated at home by mother.	Sedatives. Antitoxin, intraspinally, 10,000 units; subcutaneously, 45,000 units. Total antitoxin, 55,000 units in 2 days.	Died, Oct. 5, '10 in convulsion	Frazier	Ashhurst and Griffith.
19	Feb. 4, '11	6, M.	Hand	Dec. 23, '10. Laceration. Fall on icy street.	9 days	4 days	Severe. Convulsions.	Sutured.	Sedatives, and magn. sulph. intraspinally. Antitoxin, intraspinally, 9500 units; subcutaneously, 10,000 units. Total antitoxin, 19,500 units in 2 days.	Died, Jan. 7, '11 in perfect relaxation	Frazier	Ashhurst and Johnston.
20	Feb. 4, '11	27, F.	Uterus	Jan. 9, '11. Instrumental delivery.	Less than 22 days	4 days	Mild onset. Chronic. No convulsions.	Uterus packed for secondary hemorrhage Jan. 19, '11.	Sedatives, chloretone Phenol, 3 per cent. sol. for 1 day. Antitoxin, subcutaneously 26,500 units in 10 days.	Recovered, but died 9 days later (Feb. 22, '11) of puerperal sepsis	Mutschler	Mutschler and Johnston.
21	Apr. 17, '11	18, M.	Fingers	Apr. 10, '11. Crush by machine.	7 days	Few hours	Severe. No convulsions.	Fingers amputated.	Abscesses drained. Sedative, morphine only. Antitoxin subcutaneously, 30,000 units in 4 days.	Died, Apr. 19, '11	Neilson	Alexander and Campbell.
22	Oct. 3, '11	18, M.	Scrotum	Sept. 23, '11. Punctured wound 3 inches deep from fall on rusty iron.	10 days	Few hours	Very severe. Convulsions.	No treatment.	Sedatives, and magn. sulph. intraspinally. Antitoxin subcutaneously, 59,000 units in 4 days.	Died, Oct. 7, '11	Frazier	MacFarland
23	Mar. 15, '12	28, M.	Foot	Mar. 5, '12. Punctured wound by brass bolt.	8 days	2 days	Slow onset. Severe. No convulsions.	Bandaged. No antiseptic treatment.	Sedatives. Antitoxin, subcutaneously, 224,000 units in 3 days.	Recovered, Apr. 3, '12	Neilson	John.

ANALYSIS OF EPISCOPAL HOSPITAL CASES.

Twenty-three cases. Mortality, 56.5 per cent.
10 recovered (3, 6, 8, 9, 10, 11, 14, 15, 20, 23).
13 died (1, 2, 4, 5, 7, 12, 13, 16, 17, 18, 19, 21, 22).
Incubation under ten days, 11 cases. Mortality, 63.6 per cent.
4 recovered (3, 6, 10, 23).
7 died (2, 7, 13, 16, 19, 21, 22).
Incubation over ten days, 12 cases. Mortality, 50 per cent.
6 recovered (8, 9, 11, 14, 15, 20).
6 died (1, 4, 5, 12, 17, 18).
Efficient treatment within twelve hours of symptoms, 5 cases. Mortality, 20 per cent.
4 recovered (9, 10, mild cases; 11, 23, severe cases).
1 died (17, severe case).
No efficient treatment within twelve hours of symptoms, 18 cases. Mortality, 66.6 per cent.
6 recovered (3, 6, 8, 14, 15, 20, all mild cases).
12 died (1, 2, 4, 7, 12, 13, 16, 18, 19, 21, 22, severe cases; 5, mild case).
Less severe type of disease, 9 cases. Mortality, 11 per cent.
8 recovered (3, 6, 8, 9, 10, 14, 15, 20).
1 died (5).
Very severe type of disease, 14 cases. Mortality, 85.7 per cent.
2 recovered (11, 23).
12 died (1, 2, 4, 7, 12, 13, 16, 17, 18, 19, 21, 22).
Wounds of upper extremity, 9 cases. Mortality, 89 per cent.
Wounds of lower extremity, 8 cases. Mortality, 50 per cent.
Wounds of trunk, 3 cases. Mortality, 33.3 per cent
Wounds of head, 3 cases. Mortality, 0.0 per cent.
Antitoxin used in all 23 cases.
Efficiently as to method and quantity in 12 cases. Mortality, 46.1 per cent.
7 recovered (3, 8, 9, 10, 11, 15, 23).
5 died (13, 16, 17, 18, 19).
Inefficiently in 11 cases. Mortality, 72.7 per cent.
3 recovered (6, 14, 20).
8 died (1, 2, 4, 5, 7, 12, 21, 22).

CASE HISTORIES.

CASE 1.—Edward C., aged twenty-three years. Admitted, October 9, 1905. Discharged, October 10, 1905. Died. Service of Dr. Sinkler. Attending, Drs. Sinkler and Sweeney.

Left arm was vaccinated September 18; it began to get sore in four days, and patient had slight nausea. Then felt well until October 7 (nineteen days after vaccination), when he had head-

ache, pain in back of neck, nausea, and vomiting, with beginning trismus. Family physician dressed arm and advised removal to hospital, but patient stayed home two days longer.

On Admission (third day after first symptoms) there was trismus and retraction of head, abdomen scaphoid and rigid. On arm a slough the size of a silver dollar, baring muscle, which is black and sloughing and surrounded by area of necrotic fat; no pus.

October 9. On admission, given morph. sulph., gr. ¼, at 9 A.M., 4 P.M., and 9 P.M.; tetanus antitoxin, 20 c.c. in morning, repeated in afternoon, hypodermically. At 6 P.M. there was suggestion of a convulsion.

October 10. Convulsion at 6 A.M. for four minutes. Legs rigid, slight opisthotonos; spasms at approach of anyone. Given potass. bromide, gr. xl, and chloral hydrate, gr. xxx, by enema; also morph. sulph. at 3 A.M. and 4 A.M., hypodermically. Also 20 c.c. of antitoxin. *Died*, 8 A.M.

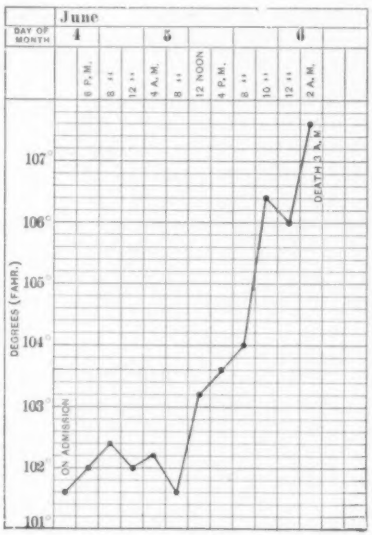


FIG. 1.—Typical temperature chart of fatal case. (Case 2.)

CASE 2.—George V., schoolboy (age unknown), under twelve years. Admitted, June 4, 1906. Discharged, June 6, 1906. Died. Service of Dr. Neilson. Attending, Dr. Owen.

On May 26, was kicked by a horse, sustaining green-stick fracture of left forearm, with lacerations of skin; not a compound fracture. Treated in surgical dispensary for the next week. On June 2 felt ill, went to bed, but was up and about the next day.

On June 4 complained of *bad pain in affected arm*, trismus and headache; had one or two slight convulsions, and was sent to the hospital

On Admission (same day as first symptoms). Jaws could be opened one-half inch; neck was stiff, abdomen rigid, legs stiff, but not rigid. Knee-jerks present on both sides, also ankle clonus. Is able to talk, has no pain until disturbed, then becomes temporarily rigid.

June 4. On admission, given bromide, gr. xv, and chloral hydrate, gr. v, every four hours by rectum. Given 30 c.c. of tetanus antitoxin every three or four hours, hypodermically.

June 5. Dose of tetanus antitoxin increased to 60 c.c. every four hours hypodermically. Given in all 330 c.c. of antitoxin hypodermically.

June 6. Given 10 c.c. of antitoxin (1500 units) intraspinally, at 12.30 A.M. *Died* at 3.15 A.M., "evidently from sudden paralysis of respiratory centres."

CASE 3.—Steven C., aged forty-eight years. Admitted, August 6, 1906. Discharged, September 2, 1906. Recovered. Service of Dr. Deaver. Attending, Drs. Weber and Aufhammer.

Right hand caught in machinery on morning of admission; diagnosis on admission, compound fracture of wrist-joint, badly lacerated and contused. Wound did fairly well.

August 15. (Nine days after injury.) Developed trismus and some stiffness of muscles of neck.

August 16 Given sodium bromide, gr. x, and chloral hydrate, gr. x, every six hours; also tetanus antitoxin, 30 c.c. every two hours hypodermically.

August 17. Given sodium bromide, gr. xv, and chloral, gr. lx, every six hours; also antitoxin as before.

August 19. Given sodium bromide, gr. xx, and chloral, gr. x, every six hours.

August 20. Sodium bromide, gr. xv, and chloral, gr. x, every six hours. Same treatment continued until

August 23. When patient developed delirium tremens, pneumonia, and later empyema. Transferred to surgical ward. *Cured of tetanus*, September 2, 1906. *Died* after many months as result of empyema.

CASE 4.—James McD., aged forty-four years. Admitted, March 5, 1908. Discharged, March 6, 1908. Service of Dr. Davis. Attending, Drs. Davis and Brown. Died.

Eight weeks ago, while chopping down a tree, patient lacerated his right great toe with an axe. Came to surgical dispensary five weeks later (three weeks ago). On March 2, began to have pain in back; pain and stiffness increased.

March 3. Neck and jaw became stiff. This morning the jaw became fixed. No convulsions. Walked to hospital.

On Admission. Jaws can be opened one-quarter of an inch; muscles stiff and spastic. Dr. Davis amputated the toe under ether. Patient had a convulsion during the operation.

Antitoxin, 3000 units, at 6 P.M., subcutaneously; antitoxin, 3000 units, at 10.30 P.M., subcutaneously.

March 6. Antitoxin, 3000 units, at 4 A.M., subcutaneously; antitoxin, 3000 units, at 9 A.M., subcutaneously. At 10 A.M. the patient *died of asphyxia in convulsions.*

CASE 5.—Katie M., aged twenty-six years. Admitted, May 31, 1908, at 12.30 A.M. Discharged, June 30, 1908, 5.35 P.M. Died. Service of Dr. Neilson. Attending, Drs. Neilson and Price.

Admitted for compound fracture of left tibia and fibula, simple fracture of right tibia and fibula. Injured by explosion in naphtha launch on the Delaware River, all sixteen passengers being thrown into the river. Patient badly shocked.

June 20. For some days has had slight spasticity of facial muscles and tendency to sardonic grin, more noticeable in the morning; some stiffness of muscles at back of neck; no convulsions. Sodium bromide, gr. xx, and chloral, gr. v, every four hours.

June 21. Jaws, back, and neck still stiff; facial muscles, better.

June 22. Trismus continues, but is no worse; swallows easily; says she bites her tongue whenever she sleeps.

June 23. Patient not so well; jaws stiffer; twitches more; difficulty in swallowing; neck stiff and painful. Antitoxin, 3000 units, twice (6000 in all, two hours apart). Sodium bromide, gr. xx, and chloral hydrate, gr. x, every four hours. Morph. sulph., gr. $\frac{1}{8}$, and atropine sulphate, gr. $\frac{1}{150}$, every six hours.

June 24. Antitoxin, 3000, t. i. d. (9000). Has a slight general convulsion each time disturbed.

June 25. Temperature, pulse, and respiration, rising. Temperature, 106.4° F.; comatose; sedatives stopped; sponged, p. r. n.

June 26. Conscious and restless; sedatives again.

June 27. Less restless. Antitoxin, 3000 units, at 5.30 P.M.

June 28. Comatose; Cheyne-Stokes respiration.

June 29. Comatose; did not move all day.

June 30. Comatose; died at 5.35 P.M.

CASE 6.—Leslie R. E., aged thirteen years. Admitted, August 12, 1908. Discharged, August 23, 1908. Recovered. Service of Dr. Deaver. Attending, Dr. Corson.

Six days before admission was struck behind the right ear with a rusty knife, which stuck in his neck; this penetrated about one and one-half inches, and was withdrawn with difficulty; small wound (punctured by thin blade); taken to another hospital where suture was put in wound and no attempt at drainage made. Began to feel stiffness at back of neck, headache and fever, and twitching of face muscles four days later (two days before admission).

On Admission. Small crust over wound behind right ear, with one silkworm gut suture; this was removed, wound opened and packed. Antitoxin, 1500 units, hypodermically.

August 13. Still slight stiffness of jaws. Antitoxin, 1500 units, hypodermically.

August 14. All muscular stiffness gone.

August 20. Out of bed.

August 23. Discharged. Diagnosis of tetanus considered positive at the time in the opinion of Dr. George Thomas (family physician) and Dr. Deaver.

November 17, 1912. Returned for examination. Has had no symptoms since. In perfect health. Small cicatrix, scarcely visible, over base of right mastoid process.

CASE 7.—Andrew H., aged thirty-five years. Admitted, August 28, 1908. Discharged, August 30, 1908. Died. Service of Dr. Deaver. Attending, Drs. Deaver and Corson.

Ran a rusty nail into his foot on August 24; came to the dispensary on August 27, because foot swelled up. Wound was opened "clear through," but is still painful, and is kept in the ward because he lives at a distance.

August 29. At 4 P.M. the patient suddenly developed stiffness of jaws and back of neck. Very much excited and irritable. Foot laid freely open where the nail had penetrated. Carbolized and packed. Antitoxin, 2000 units, subcutaneously. Sodium bromide, gr. xx, and chloral, gr. x, every six hours.

August 30. More opisthotonos; very restless. Antitoxin, 3000 units, hypodermically. *Died* at 10 P.M.

CASE 8.—Joseph G., aged thirty-four years. Admitted, September 28, 1908. Discharged, November 3, 1908. Recovered. Service of Dr. Frazier. Attending, Drs. Ashhurst and Corson.

Four days before admission was thrown from a wagon and was dragged, receiving a brush burn of the abdomen. Referred to ward as recent accident, because of hematoma of the left thigh.

October 6. Hematoma incised.

October 7. Jaws stiff. Antitoxin, 1500 units, subcutaneously.

October 8. Jaws still stiff; no pain except when he eats; a little twitching at night.

October 9. Stiffness in abdomen and across lower back. Jaws can be opened one-half inch. Headache all night. Antitoxin, 1500 units. Transferred to isolation ward.

October 10. At 2.30 A.M. had convulsion, tetanic in type. Threw his head back and had opisthotonos. Given chloroform and 4 c.c. of slightly turbid cerebrospinal fluid withdrawn and 3000 units of antitoxin injected. Brush burn curetted. Sodium bromide, gr. xx, and chloral, gr. xx, every six hours; 9 A.M., patient rather delirious; a little nauseated; back of neck stiff. Antitoxin, 3000

units, every four hours, subcutaneously. (Stopped October 17.) 10 A.M., jaws open one-quarter inch; some pain in head.

October 12. Headache ceased.

October 13. Abdomen much less rigid. Neck limber and back much less rigid.

October 14. Jaws open one inch.

October 17. Antitoxin reduced to 1500 units, every four hours. Sedatives decreased.

October 21. Slightly comatose.

October 24. Abdomen soft.

October 31. Sent back to ward as recovered.

CASE 9.—Thomas W., aged fifty-five years. Admitted, September 23, 1908. Discharged, December 5, 1908. Recovered. Service of Dr. Frazier. Attending, Drs. Ashhurst and Aufhammer.

A gunshot wound of the right temporal region, with a fracture of the orbit and rupture of the eyeball. Dr. G. O. Ring in consultation for the ocular condition.

October 20. Four weeks after injury. Complains of stiffness of jaws, says it started October 19; 1500 units antitoxin, hypodermically. Sodium bromide, gr. xx, every six hours. Transferred to isolation ward.

October 21. Antitoxin, 1500 units, every four hours.

October 23. Antitoxin, 1500 units, every eight hours.

October 24. Antitoxin, 1500 units, every ten hours.

October 26. Antitoxin, 1500 units, every twelve hours.

October 27. Antitoxin, 1500 units, every sixteen hours. Cured of tetanus, October 27, and transferred to convalescent ward, where Dr. Ring enucleated eyeball on November 17.

CASE 10.—John S., aged forty-nine years. Admitted, November 12, 1908. Discharged, December 8, 1908. Recovered. Service of Dr. Frazier. Attending, Drs. Ashhurst and Gracey.

Chief complaint. Stiffness of jaws and sore foot.

November 4. The patient ran a rusty nail through the shoe into his foot. Pulled the nail out and washed the foot with chloride of lime and soda ash and put ham fat on it. The foot began to swell, and he used iodine and arnica. One week after injury to the foot jaws began to stiffen so that he could not eat. Came to the dispensary and was sent to the ward November 12.

On Admission. Jaws can be opened one-half inch; right leg is rigid and painful. Has had spasms of jaws, which snap shut and cannot be opened.

November 12. At 7 P.M., under chloroform, 3000 units of tetanus antitoxin was injected into the subdural space of the cord; the foot, which meanwhile had healed, was reopened through the plantar fascia widely, and 1500 units of antitoxin injected deeply into muscles of foot; 1500 units of antitoxin injected, every four hours, hypodermically for five doses.

Summary (until morning of November 14). First day: Antitoxin intraspinally, 3000 units; into wound, 1500 units; subcutaneously, 7500 units. Total 12,000 units. Calomel, gr. ss.; mag. sulph., oz. j; whisky, fl. oz. iij; chloral, gr. lx; potassium bromide, gr. cxx.

November 14 and 15. Second day: Antitoxin, 13,500 units, subcutaneously; chloral, gr. lx; potassium bromide, gr. clx; whisky, fl. oz. vii; calomel, gr. j; mag. sulph., oz. j.

November 15. Third day: Antitoxin, 4500 units, subcutaneously; potassium bromide, gr. lxxx; whisky, fl. oz. vss.

November 16. Fourth day: No antitoxin. No sedatives. Patient transferred to men's medical ward for bronchitis.

November 26. Patient given 3000 units of antitoxin hypodermically for pain in jaws. No further symptoms of tetanus.

November 17, 1912. Returns for examination, four years after recovery. Says for two years right foot and leg troubled him, being sometimes weak, dragging as he walked, but at other times had no trouble. In December, 1910, on stepping suddenly off a trolley car, right leg became spastic, jaws locked, and patient had to grasp a street post to prevent falling. Under great muscular effort he managed after several minutes to open his mouth. No such attack since; but occasionally had shooting pains in leg and up the spine to the head. Has been under medical treatment for the past year, and has had no trouble with his leg during that time. Until the last year has had to be laid off work for several weeks at a time once or twice annually. Physical examination is negative. Linear, supple scar on sole of right foot, two inches long. Knee-jerks normal. No paresis or spasticity.

Case 11.—Thomas C., aged nineteen years. Admitted, December 12, 1908. Discharged, December 31, 1908. Recovered. Service of Dr. Frazier. Attending, Drs. Ashhurst and Gracey.

November 30. While running along floor of mill, soaked in machine oil, where he works, a large splinter ran through a hole in his shoe and penetrated the sole of his left foot near the head of the metatarsal of the great toe. The patient went to the dispensary, where the wound was cauterized and drained. Second visit to the dispensary two days later, but made no further visits, the patient himself removing catgut drainage that had been introduced. Was away from his work for one week.

December 17. The eighteenth day after the injury. Patient did not feel well and his jaws were sore and stiff. Took to his bed on December 18. That night he says his back muscles contracted until he rested only on the back of his head and heels. Was sent to the hospital by family physician who saw him in the morning.

On Admission. Jaws can be opened one-half inch; considerable stiffness of muscles of the back of neck and some stiffness of the back muscles; abdomen markedly rigid and a tendency to stiffness

of the legs. Wound on foot has counteropening for drainage, two inches distant. As soon as possible after admission patient was given chloroform and 3000 units of antitoxin injected intraspinally; the wound in the foot was opened deeply through the plantar fascia, scraped out and packed with gauze soaked in iodine, 1 to 3 of water. Three splinters removed from the wound. Culture on blood serum and an anaërobic culture made (streptococci; no Bacillus tetani).

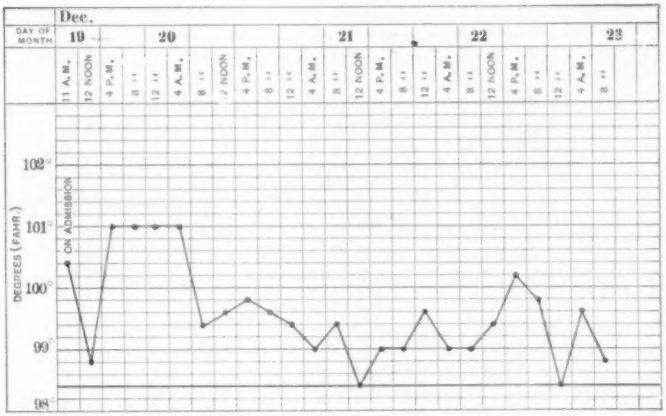


FIG. 2.—Typical temperature chart of severe case arrested promptly with ultimate recovery. (Case 11.)

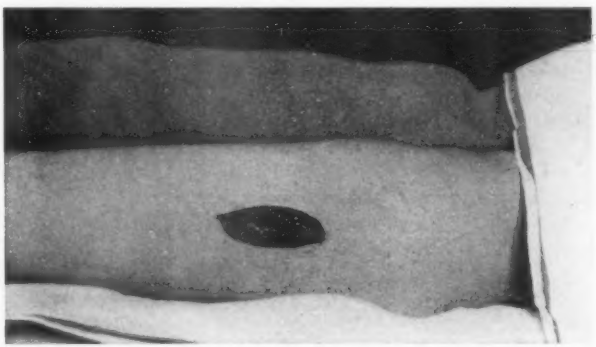


FIG. 3.—Granulating wound through which antitoxin had been injected into the sciatic nerve. Wound left open for subsequent injections, which, however, were not necessary. (Case 11.)

December 20. Jaws opened one and one-eighth inches; not so stiff in the neck muscles. Chloral hydrate, gr. x, and potassium bromide, gr. xx, every three hours. Total chloral hydrate, gr. lx,

and potassium bromide, gr. cxx. At 3.30 P.M., 750 units of antitoxin was injected into the sciatic nerve, which was exposed by an incision under chloroform, and 750 units injected around the wound in the foot.

December 21. Jaws opened one and three-quarter inches. Summary: No antitoxin; chloral hydrate, gr. c; potassium bromide, gr. clx.

December 22. Summary: No antitoxin; chloral hydrate, gr. lxxx; potassium bromide, gr. clx.

December 23. Summary: No antitoxin; chloral hydrate, gr. l; potassium bromide, gr. c.

December 24. Summary: No antitoxin; chloral, gr. xxxv; potassium bromide, gr. lxx.

December 25. Summary: No antitoxin; chloral, gr. xx; potassium bromide, gr. l. Patient is now convalescent. Chloral and bromides continued in diminishing doses for three days longer.

December 31. Patient transferred to surgical ward as cured of tetanus.

CASE 12.—Francis C., aged sixty-three years. Admitted, October 16, 1909. Discharged, October 19, 1909. Died. Service of Dr. Frazier. Attending, Drs. Ashhurst and Hopper.

September 28. Fingers of the right hand badly lacerated in a picker in a woollen mill. Patient treated in the dispensary and attending daily.

October 15. Patient felt jaws becoming stiff and sent for the ambulance twelve hours later.

On Admission. October 16. General physique poor; jaws stiff; opisthotonos; abdominal muscles board-like; neck retracted; reflexes, especially the patellar, markedly increased. Chloral, gr. x, and sodium bromide, gr. xx, every two hours.

October 17. General condition poor. Under chloroform lumbar puncture was made and 1500 units of antitoxin injected. All the fingers of the right hand amputated at the metacarpal joint; even under chloroform the marked lordosis could not be made to disappear. Chloretone, gr. xxx, every two hours. Stopped the sodium bromide and chloral.

October 18. Patient semicomatose; heart bad. P.M. Heart weakening. No increase of rigidity.

October 19. Patient died at 4 A.M.

CASE 13.—Margaret D., aged eleven years. Admitted, November 20, 1909. Discharged, November 22, 1909. Died. Service of Dr. Frazier. Attending, Drs. Ashhurst and Hopper.

November 11. Patient fell and abraded right knee. Was treated at home and became infected. Jaws became stiff in the morning of November 19, but went to school and on return again called mother's attention to her stiff jaws. Went to bed and slept well. In the morning (November 20) jaws were rigidly clenched, and

when she tried to move or be moved, she became very rigid; she had pain in jaws, neck, and back. Her family physician saw her, said she had symptoms of lockjaw, left some medicine and asked to be called on the phone in the evening. When he heard her condition then he sent a note asking for the ambulance and her admission to the hospital.

FIG. 4.—Opisthotonos. Death six hours later in convulsions. (Case 18.)

On Admission (November 20, 10.30 P.M.). Jaws were set, head retracted, legs extended and rigid; moderate lordosis; board-like scaphoid abdomen; mind clear; reflexes all increased; suppurative abrasion over tubercle of the right tibia covered with a bread poultice.

November 21, 12.30 A.M. (two hours after admission). Patient was operated upon (chloroformed.) The anterior crural and obturator and sciatic nerves were exposed and 750 units of antitoxin injected into each of anterior crural and obturator and 1500 units into sciatic; about 1500 units injected around wound in the leg, after disinfecting it and swabbing it out with 3 per cent. alcoholic solution of iodine. Continuous saline proctoclysis. Chloretone, gr. xv, every four hours by mouth. Chloral hydrate, gr. xx, and sodium bromide, gr. xxx, every two hours by rectum in saline proctoclysis. During the morning (November 21) the patient was quiet, jaws slightly less rigid and can now open one-quarter of an inch. Seems better generally.

2.30 P.M. Incisions reopened and same dose of antitoxin injected into anterior crural, obturator, and sciatic nerves; 1500 units of antitoxin given intraspinally under chloroform.

4.30 P.M. Teeth can be separated further.

7.30 P.M. Patient not so well. Temperature, pulse, and respiration all rising.

10.30 P.M. Patient restless. Jaws still less rigid. Clonic contractions of muscles of back and legs every few minutes; 1500 units of antitoxin hypodermically.

November 22, 1.30 A. M. No change.

9.30 A.M. Patient weaker. Chloretone stopped. Tincture of digitalis, minims v. Camphorated oil, minims xx, every four hours.

12.00 M. Antitoxin as above into nerves under chloroform; none given intraspinally.

3.00 P.M. Patient has been comatose ever since chloroform this noon. Respirations shallow and labored. Pulse better.

6.30 P.M. Patient died, in relaxation.

CASE 14.—Sarah McV., aged forty-five years. Admitted, February 4, 1910. Discharged, February 19, 1910. Recovered. Service of Dr. E. J. Morris. Attending, Drs. Ashhurst and Siner.

January 23 (twelve days before admission). When pregnant three months, fell down stairs in bare feet and sprained back and received small lacerated wound between fourth and fifth right toes; no other injuries. The interdigital cleft became very painful the next day and the foot began to swell. Patient treated the foot at home for ten days and then on February 2, came to the surgical dispensary and was dressed. Returned two days later and complained of pain on opening mouth and stiffness of jaws. Referred at once to isolation by Dr. Ashhurst, after renewed antiseptic treatment of wound with iodine (3 per cent.).

On Admission, February 4. Chief complaint: Stiffness of jaws and neck and wound of right foot.

Examination. Reflexes slightly increased; no ankle clonus or Babinski; abdomen not rigid; patient three months pregnant; 3000 units of antitoxin subcutaneously on admission. Potassium bromide, gr. xxx, and chloral hydrate, gr. xv, every four hours. Mag. sulph., oz. ss.

February 5. Patient complains of headache, backache, and muscular pains all over; abdomen somewhat tender and rigid. Reflexes slightly increased; 1500 units of antitoxin, one-half around wound in foot and one-half in abdominal wall, hypodermically. Bromides and chloral only t. i. d.

February 6. Pains in pelvis; uterine bleeding all day, passing several large clots.

Pelvic examination: Large soft cervix; fundus above pubis; os not dilated.

February 7. At 2 A.M. a very profuse metrorrhagia. Os now patulous and placenta protruding. Uterus cleaned out with placental forceps and packed. Fluid extract of viburnum prunifolium, dram j every three hours.

10.00 A.M. Patient in good condition.

February 8. Packs removed; 1500 units of antitoxin subcutaneously.

February 19. Patient has done well. Discharged today as cured of tetanus.

CASE 15—Mary S., negro, aged twenty-three years. Admitted, February 21, 1910. Discharged, March 8, 1910. Recovered. Service of Dr. Neilson. Attending, Drs. Ashhurst and Siner.

January 26, at 2 A.M., while walking on the street, the patient was struck on the head by a brick, causing a slight laceration. She was taken in the ambulance to another hospital, where the laceration was sutured and the patient discharged one hour later. Returned on the tenth day and had the sutures removed. She says that at that time there was a small tender swelling in the wound.

February 14. One week before admission, and nineteen days after the wound, the patient's jaws gradually became painful, and she began to have difficulty in opening her mouth. The condition gradually grew worse, and she can now barely separate her jaws one-quarter of an inch, with much pain.

On Admission, February 21. Temperature, 100° F. Chief complaint is stiffness of jaws. Postauricular lymph nodes on right enlarged. Cicatrix from recent wound on scalp, near inion; below is a small, tender lump. Abdomen a little tender. Some rigidity of muscles of back and neck. Patellar reflexes greatly exaggerated on the right and increased on the left. No ankle clonus or Babinski. Mag. sulph., oz. ss., stat. Antitoxin, 3000 units subcutaneously on admission and 1500 units intraspinally. Potassium bromide, gr. xxx, and chloral hydrate, gr. vii, every three hours by mouth. Hyoscine, gr. $\frac{1}{100}$, hypodermically, 3 P.M. and 11 P.M. Abscess on scalp opened.

February 22. Antitoxin 3000 units subcutaneously at 10 A.M. Sedatives as before. Patient's condition not so good. Jaws nearly closed. Neck and back somewhat rigid. Stuporous, and mind wanders. More pain in head. Antitoxin, 3000 units, at 4 P.M., subcutaneously; 1500 units at 8 P.M., when there was some improvement. Jaws not so rigid.

February 23. 10 A.M., 2 P.M., and 8 P.M., 1500 units of antitoxin subcutaneously. Condition the same. Morphine, gr. $\frac{1}{8}$. Great pain, worse in the back. Sedatives as before.

February 24, A.M., and P.M., 1500 units of antitoxin subcutaneously. Jaws opened one-half inch in the morning, and open one inch by evening. Sedatives as before.

February 25. No more sedatives after February 24; 1500 units of antitoxin subcutaneously. Tincture of digitalis, minims x, t. i. d.

February 26, 3000 units of antitoxin subcutaneously. Pains in lower jaw.

February 27. No antitoxin after this date.

February 28. Patient can flex neck until chin is within two inches of chest.

March 1. Can make chin touch chest.

March 2. Sat up in bed for first time. Soft diet for first time.

March 3. In chair for one-half hour.

March 4. Jaws open three-eighths of an inch.

March 5. In chair for two hours.

March 6. Patient walked a little.

March 7. Jaws can be opened one-half inch. Neck still somewhat rigid, and when it is flexed until chin touches sternum she has some pain in the back.

March 8. Discharged as cured.

CASE 16.—Charles M., aged eight years. Admitted, July 17, 1910. Discharged, July 20, 1910. Died. Service of Dr. Deaver. Attending, Dr. Griffith.

July 5. Was cut on right thumb by hatchet. Wound was dressed in dispensary, using three sutures. No symptoms until Friday evening, July 15, when he complained of a little difficulty in swallowing. On July 16, felt all right. On July 17, before admission, had a spasm which lasted only a few minutes.

On Admission. Patient perfectly relaxed, with the exception of some slight stiffness of jaws, and a sardonic grin, which was marked. Given 43,000 units of antitoxin hypodermically. Chloral, gr. ij, and potassium bromide, gr. vi, by mouth, every three hours. Had three spasms on day of admission, each lasting about three minutes, after which he was relaxed.

July 18. Temperature higher. General condition not so good. Better toward night. Five spasms during the day, each about three minutes, one severe. Opisthotonos marked in last convulsions and relaxation afterward not so complete. Sweating is marked; 5000 units of antitoxin, every three hours, for eight doses—40,000 units, hypodermically. Bromide and chloral as before.

July 19. General condition good, but jaws more locked. Some difficulty in swallowing. Pain in abdomen. Cyanosis of lips marked during spasms. Four spasms today, one quite severe; 20,000 units of antitoxin.

July 20. General condition not so good. Jaws locked. Took a bad turn at 9 P.M. Temperature, 106° F. Died at 10.30 P.M.

CASE 17.—Harry F., aged seven years. Admitted, October 14, 1910. Discharged, October 16, 1910. Died. Service of Dr. Frazier. Attending, Dr. Henneberger.

Three weeks ago the patient was vaccinated, and was all right until the evening of October 13, when malaise was noted and he complained of stiffness of back and soreness in jaws. Brought to the hospital the next morning.

Examination. Jaws somewhat stiff and unable to open mouth to full extent. Abdomen scaphoid, with great rigidity of abdominal muscles. The least tapping throws them into a tetanic contraction; 5000 units of antitoxin, every four hours, subcutaneously. Mag. sulph. (intraspinally), 2 c.c. of 25 per cent. solution at 12.30 A.M., October 15.

October 16. Died at 1 A.M. Temperature, 104° F. Total amount of antitoxin in two days, 60,000 units subcutaneously.

CASE 18.—Minnie P., aged six years. Admitted, October 3, 1910. Discharged, October 5, 1910. Died. Service of Dr. Frazier. Attending, Drs. Ashhurst and Griffith.

September 7. Patient was vaccinated. She did well. Scab formed, which was knocked off at play and no other scab formed. Mother dressed the wound and a slight infection occurred, but healing proceeded normally from outside toward the centre. No untoward occurrence until October 1, when the child complained of mouth being sore, but was able to eat and play and slept well the night of October 1. Complained of mouth all of October 2, but could swallow, and went to Sunday school. On Monday, October 3, had malaise, and a weak spell in which she fell and received an ugly contusion on forehead. During the day she still complained of mouth being sore and some slight stiffness. General muscular rigidity noticed, and jaws were somewhat hard to open. Brought to the hospital on the night of October 3.

On Admission. Contusion on the forehead the size of an orange, received by a fall in a weak spell. Opisthotonos; sardonic grin marked; angles of mouth drawn down; muscular rigidity marked; complains of pain in stomach; head retracted; had one slight convulsion before admission; sent to isolation ward, October 4; 5000 units of antitoxin subcutaneously on admission.

October 4. Muscular rigidity marked; opisthotonos and sardonic grin marked. Patient able to swallow. Had about two convulsions an hour today, but very slight. Potassium bromide, gr. lx, and chloral, gr. xxx, every three hours; 25,000 units of antitoxin subcutaneously.

October 5. Seen by Dr. Ashhurst on this date first; 10,000 units of antitoxin intraspinally, and 15,000 units of antitoxin subcutaneously. Temperature rising. Convulsions increasing in severity. Patient is very restless, but able to take nourishment and medicine. A very severe convulsion at 3 P.M., followed by two slighter ones. Temperature, 106° F. At 4 P.M. several slight convulsions; breathing very labored; cold, clammy sweat. At 5.30 P.M. had three very severe convulsions, and died at 5.45 P.M. Temperature, 106.6° F.

CASE 19—Thomas B., aged six years. Admitted, January 4, 1911. Discharged, January 7, 1911. Died. Service of Dr. Frazier. Attending, Drs. Ashhurst and Johnston.

Patient has a tapeworm, and has lost weight in the last few months. On December 23, while running in the street, he fell over a Belgian block, striking the palm of the right hand against a sharp piece of ice. A U-shaped laceration at the thenar eminence resulted, which bled very profusely. Was taken to another hospital, where two sutures were inserted, which controlled the hemorrhage. No antitoxin was given.

January 1. It was first noticed that his jaw was stiff and that he spoke like a child who was tongue-tied. He was taken back to the same hospital, where, on account of the difficulty in opening the mouth and some patches on the tongue, diphtheria was suspected, but by mouth gag no membrane was found in the throat.

January 4. A positive diagnosis of tetanus was made, and he was sent to the Episcopal Hospital.

On Admission. Teeth cannot be separated more than one-half inch. Face is wrinkled and angles of the mouth are drawn down and out in a sardonic grin. Depressors of the jaw become tense on any attempt to open mouth. When the patient is quiet, nothing abnormal is apparent. Neck muscles and those of the back are a little stiff, but no opisthotonos is present. Limbs are normal. A clean granulating wound on the right thenar eminence. 12 midnight; 5000 units of antitoxin in right forearm.

January 5, 4.00 A.M. Chloral hydrate, gr. iij.

6.00 A.M. Slight convulsion.

8.00 A.M. Jaws more rigid.

9.00 A.M. 5000 units of antitoxin intraspinally.

1.30 P.M. Potassium bromide, gr. v. Weaker. Delirious. Temperature, 104° F.

4.00 P.M. Chloral hydrate, gr. iij. Patient sleeping.

8.00 P.M. 5000 units of antitoxin subcutaneously. Potassium bromide, gr. v.

12.00 P.M. Chloral, gr. iij. Temperature, 103.4° F.

January 6, 4.00 A.M. Potassium bromide, gr. v. Temperature, 100.2° F.

12.00 M. 4500 units of antitoxin intraspinally. Seen by Dr. Ashhurst on this date first.

1.00 P.M. Potassium bromide, gr. v. Temperature, 101.8° F. Chloral, gr. iiss by rectum.

4.00 P.M. Sleeping quietly.

8.00 P.M. Potassium bromide, gr. v, and chloral, gr. ijss by rectum, which was expelled.

9.00 P.M. Potassium bromide, gr. v, and chloretone, gr. v.

11.00 P.M. Sleeping.

January 7, 1.00 A.M. Chloretone, gr. v, because he awoke.

3.00 A.M. Potassium bromide, gr. v. Temperature, 100.4° F.

9.00 A.M. Potassium bromide, gr. v.

12.00 M. Temperature, 101° F.

2.00 P.M. Mag. sulph., $3\frac{3}{4}$ grams in a 25 per cent. solution intraspinally (7 c.c. of solution, clearly an overdose).

3.00 P.M. Patient died in perfect relaxation. Probably would have recovered if no magnesium sulphate had been given.

CASE 20.—Lillie S., aged twenty-seven years. Admitted, February 4, 1911. Discharged, February 22, 1911. Recovered. Service of Dr. Mutschler. Attending, Drs. Mutschler and Johnston.

Personal History. Has had one child. A prolonged and difficult labor with a laceration of the cervix. During the present gestation health has been good. Much better than previously.

Present Condition. January 9. While at breakfast, had a sudden profuse hemorrhage. The uterus was evacuated by her physician. Improved for ten days, when a second sudden very severe hemorrhage occurred. Two days later developed phlebitis in the left leg. Twenty-two days after delivery, four days before admission, patient began to have dull pains in muscles at back of neck, some difficulty in separating jaws, and slight pain on deglutition; all symptoms increased slowly in severity daily. On the day before admission had 5000 units of antitoxin subcutaneously.

On Admission. Able to open incisor teeth only enough to admit tip of the index finger. This causes some pain in the neck. Some rigidity of muscles of back of neck, and pain on motion. Uterus felt just above pubis.

Vaginal Examination. Small amount of brownish discharge, not especially foul. Cervix soft and lacerated; uterus enlarged, soft, not freely movable. Left leg uniformly increased in size; skin glistening, pits slightly on pressure. No pain in leg or on pressure over veins.

February 4, 6.00 P.M. Chloretone, gr. x, every four hours.

8.00 P.M. 5000 units of antitoxin in pectoral muscle. Left leg bandaged.

February 5, 8.00 A.M. Good bowel movement; appetite good; considerable thirst. Unable to open jaws enough to admit spoon.

4.00 P.M. Phenol, 3 per cent., minims, xv, every two hours, hypodermically.

8.00 P.M. Antitoxin, 5000 units, in right pectoral muscle. Patient very drowsy. Pain less in the neck.

February 6, A.M. Patient very drowsy.

4.00 P.M. Temperature, 104.8° F.; pulse, 136; respiration, 28.

5.00 P.M. All treatment stopped. Atropine sulphate, gr. $\frac{1}{100}$; morph. sulph., gr. $\frac{1}{6}$, hypodermically.

8.00 P.M. All appearances of intense sepsis. Completely relaxed. No pain anywhere. Infusion of digitalis, fl. dr., ij. Enteroclysis with normal salt solution, to take one pint every three hours; with each pint, two drams infusion of digitalis.

February 7, 12.00 A.M. Antitoxin, 5000 units, subcutaneously. Return of pain and stiffness. Chloretone, gr. x, every three hours.

February 10. 5000 units of antitoxin subcutaneously.

February 11. 5000 units of antitoxin.

February 13. 1500 units of antitoxin.

February 22. Patient gradually improved until morning of February 22, when all tetanic symptoms had been absent for some days. On latter date, she had a severe uterine hemorrhage. Uterus packed, but patient failed rapidly.

February 23, 12.15 A.M. Patient died.

CASE 21.—Poland P., aged eighteen years. Admitted, April 17, 1911. Discharged, April 19, 1911. Died. Service of Dr. Neilson. Attending, Drs. Alexander and Campbell.

April 10. While at work the patient had his hand caught in a machine; index, ring, and middle fingers cut. Middle and ring fingers amputated on the same day. Discharged on April 14. Returned to hospital on April 17, with tetanus. Jaws locked, pain in back, and stiffening of neck muscles. Hand is very much swollen, and has a very offensive odor. Stitches removed from fingers. Palm of hand opened up and one fluidounce of pus removed. Several stab wounds made on back of hand; 5000 units of antitoxin given subcutaneously every seventh hour. Hand dressed every day. Continuous 1 to 5000 bichloride dressing on hand.

April 17. Antitoxin, 10,000 units, subcutaneously; whisky, oz. j, and morph. sulph., gr. $\frac{1}{4}$.

April 18, A.M. Antitoxin, 5000 units, subcutaneously; whisky, oz. jss, and morph. sulph., gr. $\frac{1}{4}$; P.M. Antitoxin, 10,000 units, subcutaneously; whisky, oz. ij, and morph. sulph., gr. $\frac{1}{4}$.

April 19. Patient died at 3.55 A.M.

CASE 22.—John McD., aged eighteen years. Admitted, October 3, 1911. Discharged, October 7, 1911. Died. Service of Dr. Frazier. Attending, Dr. MacFarland.

September 20. The patient fell on a rusty hydrant stalk; the stem entered the scrotum, and he walked home untreated. Later on the same day he came to the surgical dispensary, where he was not treated, but referred to ward, but did not stay. No antitoxin given. Thirteen days later was admitted to the hospital with symptoms of tetanus. (How long these had existed is not stated; probably two days.)

On Admission. Well-developed adult male. Temperature, 99° F.; pulse and respiration, normal. Has a sardonic grin; head retracted; slight opisthotonos; occasional attacks of general rigidity; jaws can be opened one-half inch; over pubis in the midline is an area the size of a quarter dollar, which is tender, red, and inflamed; open wound on left side of scrotum, discharging yellow, purulent material. Probe reaches from here to area above pubis under the skin.

Treatment. Under local anesthesia, incision made over tender area at pubis, and through-and-through drainage to scrotal wound

with rubber tube. Antitoxin, 14,000 units in 5000 doses subcutaneously. Potassium bromide, dram ij, and urotropine, gr. x.

October 4. Patient is worse. Frequent attacks of spasms, and pulse and respiration have increased. Antitoxin given every six hours, to make 20,000 units, subcutaneously. Frequent convulsions, with opisthotonos lasting two to three minutes. Potassium bromide, drams ix; urotropine, gr. x; morph. sulph., gr. $\frac{1}{4}$.

October 5. Potassium bromide, drams ivss, and urotropin, gr. v. Antitoxin, 10,000 units, subcutaneously. Morph. sulph., gr. $\frac{3}{4}$; intraspinal injection of mag. sulph., 25 per cent. solution, 6 c.c.

October 6. Patient has developed pneumonia, and is in a critical condition. No convulsion since early morning. Antitoxin, 15,000 units, subcutaneously. Potassium bromide, drams ijss; urotropin, gr. xv; atropine, gr. $\frac{1}{75}$; mag. sulph., intraspinally, 3 c.c. of a 25 per cent. solution; whisky, oz. iiiss; tincture of digitalis, minims xl, hypodermically, in 10-minim doses.

October 7. Patient in very critical condition. Died at 6 P.M.

Case 23.—Gregol S., aged twenty-eight years. Admitted, March 15, 1912. Discharged, April 20, 1912. Cured. Service of Dr. Neilson. Attending, Dr. John.

Chief complaint is rigidity of jaw and neck muscles; wound of right foot.

March 5. The patient ran a piece of a brass bolt into the outer side of his foot, and went to a "lodge doctor," who bandaged it without any further treatment. Eight days later, on Wednesday, March 13, began to have pain and rigidity of muscles of jaw and neck. Was admitted on Friday night, March 15, with well-marked trismus and considerable rigidity and retraction of neck with moderate arching of the back.

On Admission. Symptoms as above. Face is drawn, mouth particularly being drawn at the corners into the typical tetanic risus sardonicus. The head is rigid and well drawn back. Complains of some pain in the neck and thorax. Is able to separate the teeth about one inch.

Thorax: Lungs negative. Heart area normal and sounds good. No murmurs. Back arched from occiput to buttocks, but the legs are freely movable. Centre of arch about three inches above the bed.

Abdomen: Board-like rigidity of all the abdominal muscles, increased on examination. A small inguinal hernia on right side.

Extremities: All are normal, with the exception of the right leg, which shows a penetrating wound on the outer side of the right foot about the middle of the arch. This runs up and back under the skin below the external malleolus for about three inches.

March 15. Wound in foot opened its whole length; slough cut away; washed with peroxide and warm boric, and packed with iodoform gauze; 6000 units tetanus antitoxin injected subcuta-

neously. Chloral hydrate, gr. xx, and potassium bromide, gr. x, every four hours, with morph. sulph., gr. $\frac{1}{8}$, hypodermically, every four hours.

March 16. 93,000 units of antitoxin injected subcutaneously. Patient is not so well. Can barely separate teeth, and has convulsive spasms every few seconds. No real convulsion, however.

March 17. 65,000 units of antitoxin injected subcutaneously. Patient can open mouth a little wider, but the convulsive spasms keep up, and there is more rigidity and arching of the back.

March 18. 60,000 units of antitoxin injected subcutaneously. Patient in about the same condition. No worse.

March 19. No antitoxin. Patient has had first real sleep this morning. Slept again for several hours in the afternoon. Convulsive spasms are not quite so frequent, nor so severe. Arching and rigidity not diminished, possibly slightly increased.

March 24. Patient is much improved, and rests quietly. Bromide and chloral reduced by half.

March 26. Patient rests quietly, but rigidity continues undiminished, and considerable urticaria over body. Given soft-boiled egg and a small piece of dry toast for dinner.

March 27. Rigidity considerably decreased, patient in good condition.

March 30. Patient's general condition is good. Rigidity is well marked but decreasing slowly; most marked in abdominal muscles. Blood pressure of left arm: systolic, 182; diastolic, 124.

March 31. Hot bath for fifteen minutes seemed to decrease rigidity to a considerable extent. Returned later in the day, but the patient was much more comfortable.

April 3. Transferred to convalescent ward.

April 7. Patient is doing well, and is up and in chair daily.

April 16. Patient put on full diet. Is up and walking about the ward. Is still a little weak, and is slightly sore in the chest, but on the whole is in good condition.

April 20. Patient is up and about. Still has slight soreness over lower thorax. Discharged in good condition.

November 17. Returned for examination. No symptoms since recovery, and in excellent health now.

References.

Almagia and Mendes. Internat. Clinics, Philadelphia, 1908, iii, 12.
Bacelli. London Med. Mag., 1893–'94, ii, 811.
Beates and Thomas. Monthly Cyclop. and Med. Bull., 1911, iv, 326.
Blake. Surg., Gyn., and Obst., 1906, i, 541.
Bockenheimer. Arch. f. klin. Chir., 1908, lxxxvi, 277.
Busch. Arch. f. klin. Chir., 1907, lxxxii, 27.
Camus. C. R. Soc. de Biol., Paris, 1912, lxxii, 109.
Dehne and Hamburger. Wien. klin. Woch., 1904, xvii, 807.
Donitz. Deutsch. med. Woch., 1897, xxiii, 428.

Fedden. Clin. Jour., 1911, xxxvii, 356, Case 2.
Fink. Jour. of Trop. Med., 1911, xiv, 161.
Fox. New York Med. Rec., 1910, ii, 720.
Frazier. Keen's Surgery, Philadelphia, 1906, i, 478.
Fricker. Deutsch. Zeit. f. Chir., 1907, lxxxviii, 429.
v. Graff. Mitth. a. d. Grenzgeb. d. Med. u. Chir., 1912, xxv, 145.
Gumprecht. Deutsch. med. Woch., 1894, xx, 546.
Gumprecht. Arch. f. d. ges. Physiol., 1894, lix, 105.
Hessert. Surg., Gyn., and Obst., 1909, ix, 145.
Hitchens. American Vet. Rev., August, 1910.
Hofmann. Beitr. z. klin. Chir., 1907, lv, 697.
Huber. Beitr. z. klin. Chir., 1912, lxxvii, 139.
Hutchings. Surg., Gyn., and Obst., 1909, ii, 11.
Imperiali. Giorn. di med. Milit., 1910, fasc. x to xi; in Policlinico, 1911, xviii, 363.
Jacob. Blumenthal and Jacob: Berl. klin. Woch., 1898, xxxv, 1079.
Jacobson and Pease. Trans. Amer. Surg. Assoc., 1906, xxiv, 254.
Kintzing. New York Med. Jour., 1911, xi, 1268.
Krauss and Amiradzibi. Zeit. f. Immunitätsforsch., 1910, vii, 1.
Küster. Centr. f. Chir., 1905, xxxiii, Beil., S. 15.
Lop. Bull. Soc. Chir., Paris, 1906, xxxii, 184.
v. Leyden. Berl. klin. Woch., 1899, xxxvi, 632; Deut. med. Woch., 1901, xxvii, 477.
Luckett. Med. and Surg. Repts., Bellevue and Allied Hosps., New York, 1904, i, 319.
Magula. Beitr. z. klin. Chir., 1911, lxxvi, 588.
Maunoury. Assoc. Franc. de Chir., 1902, xv, 608.
Marie and Morax. Ann. de l'Inst. Pasteur, 1902, xvi, 818; 1903, xvii, 335.
Matas. Trans. Amer. Surg. Assoc., 1909, xxvii, 40.
McCampbell. Jour. Amer. Med. Assoc., 1907, i, 919.
McFarland. Jour. Amer. Med. Assoc., 1903, ii, 34.
Meyer and Ransom. Arch. f. exp. Pathol. u. Pharm., 1903, xlix, 369.
Miller. Amer. Jour. Med. Sci., 1908, ii, 781.
Neary. New York State Jour. of Med., 1910, x, 476.
Nissen. Deutsch. med. Woch., 1891, xvii, 775.
Nocard. Bull. Acad. de Méd., Paris, 1895, xxxiv, 407.
Palmer. Jour. Royal Army Med. Corps, 1912, xviii, 400.
Paterson. Lancet, 1910, i, 922.
Porter and Richardson. Boston Med. and Surg. Jour., 1909, clxi, 927.
Pribram. Prag. med. Woch., 1908, xxxiii, 719.
Remertz. Inaug. Dissert., Berlin, 1911. Ueber prophyl. Injektion von Tetanus Antitoxin.
Reynier. Bull. Acad. de Méd., Paris, 1908, lix, 623, 629, 780.
Riche. Bull. et Mém. Soc. Chir., Paris, 1912, xxxviii, 476.
Rogers. Jour. Amer. Med. Assoc., 1905, ii, 12.
Roux and Borrell. Ann. de l'Inst. Pasteur, 1898, xii, 225.
Sawamura. Arbeiten a. d. Inst. z. Erforschung d. Infektionskrankh. in Bern (Kolle), Heft iv, 1, Jena, 1909.
Schnitzler. Centr. f. Bakteriol., 1893, xiii, 679.
Sewaki. Sei-i-Kwai Med. Jour., Tokyo, 1910, xxx, 33.
Simon. v. Graff: Mitth. a. d. Grenzgeb. d. Med. u. Chir., 1912, xxv, 145.
Solieri. Centr. f. Bakteriol., 1910, lv, 141.
Suter. Arch. f. klin. Chir., 1905, lxxv, 113.
Stintzing. Münch. med. Woch., 1898, xlv, 1265.
Stintzing. Mitth. a. d. Grenzgeb. d. Med. u. Chir., 1898, iii, 461.
Tarozzi. Cited. by Solieri, q. v.
Vaillard. Bull. Acad. de Méd., Paris, 1908, lix, 569, 587.
Vincent. Ann. de l'Inst. Pasteur, 1904, xviii, 748; Soc. de Biol. de Paris, in Gaz. des Hôp 1908, lxxxi, 620, 668, 921.
Zupnik. Deutsch. med. Woch., 1900, xxvi, 837.

ISOLATED SCLEROTIC INVOLVEMENT OF THE MITRAL VALVE.

By Robert N. Willson, M.D.,
of Philadelphia.

The frequency of occurrence of mitral-valve deformity and disease independent of and in the absence of other cardiac-valve implication, is one that has received attention only, as a rule, from the standpoint of vegetative endocarditis. It has been described customarily as rheumatic in origin, and at least in its acute forms has been regarded as a part of true bacterial rheumatic disease.

Sclerotic thickening and deformity, causing insufficiency or stenosis, and oftentimes both anatomic effects, have received scant attention at the hands of students of the heart, and perhaps less than has been merited by their actual frequency and interest. Both in the wards of the Philadelphia Hospital and in the autopsy opportunities of private practice I have been impressed by the sclerotic nature of occasional mitral lesions, which I had been led by the history of the case during life and its course while under my observation, to regard as subacute recrudescences of a bacterial endocarditis of a vegetative type. Usually the physical signs have indicated mitral insufficiency or mitral stenosis, or both. Less frequently the indications have been those of purely aortic disease. Irrespective of the location my attention has been attracted by the sclerotic nature of the process and the necessity of inquiring whether I had erred in my understanding of its etiology. I confess that with each new instance, in spite of the association of tonsillitis, joint symptoms, or even pericardial inflammation, I have been forced to look away from acute rheumatism as a satisfactory explanation of the lesion, and to search for a more likely immediate cause.

In this brief study I will refer only to the sclerotic changes of the mitral valve that occur in the absence of apparent lesions of the other cardiac valves. In a later communication I hope to point out the significance of similar processes limited to the aortic valve.

The Anatomical Lesion. I have already referred to the fact that I have at autopsies studied examples of sclerotic insufficiency, also of the frequent combination of sclerotic stenosis with insufficiency of the mitral valve. I have never seen such a stenosis in the absence of an accompanying anatomical insufficiency, though not always of such a character as to furnish the classical auscultatory signs. I have observed a fibrosclerotic insufficiency in the absence of a deformity amounting to an anatomical stenosis, though far less regularly did the patient during life show either the physical

signs or the symptoms of valvular obstructive disease. The photographs are illustrative of the isolated sclerotic involvement of the mitral vavles.

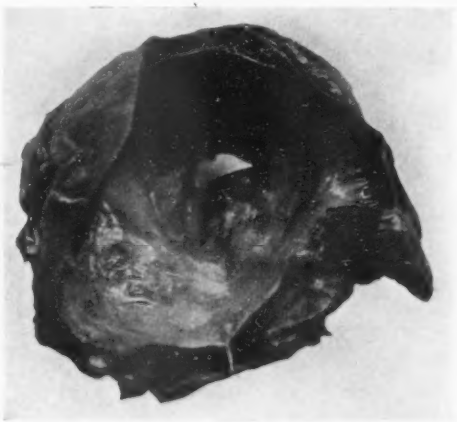


FIG. 1.—High-grade sclerotic involvement of the mitral valve resulting in stenosis and insufficiency.

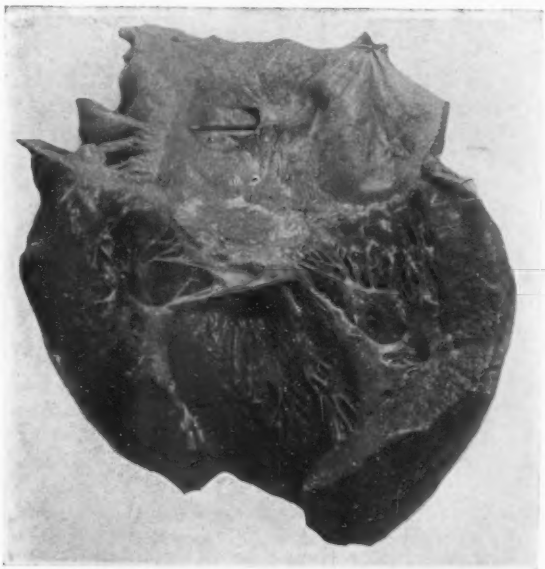


FIG. 2.—Sclerosis and atheroma of the left auricular wall and of the mitral valve.

Microscopic study of the diseased valve leaflets and cusps has shown in the early cases the typical changes of a fibrosclerosis. In more advanced cases atheromatous degeneration is often superimposed, and in a large percentage, lime salts have been deposited, and resulted in a greater or less degree of calcareous change. There has been an entire absence of bacterial findings, and, as far as staining methods are concerned, there has been a complete failure to demonstrate the presence or the causal influence of bacterial forms. I shall later refer to the possibility of the demonstrating spirochetæ in such tissue. The success of a number of laboratory workers in staining the Spirochæta pallida in sections of the aorta that have been in preservative fluids for a number of years leads us to believe that we will yet be able to determine a causal organism in at least those instances that depend upon the Spirochæta pallida for an etiologic factor. All grades of fibrosis of the mitral valve have been observed from the slightest possible thickening to a gross deformity such as is seen in Fig. 2.

THE PHYSICAL SIGNS AND CLINICAL COURSE OF THE DISEASE. I know no clinical method of distinguishing the sclerotic type of valvular disease from the vegetative except in the probable exclusion of syphilis from the etiology by the Wassermann test. In many of the cases that have come under my observation, syphilis has either been admitted by the patient, or there has been sufficient reason to believe that such an admission would be in order. I believe that many instances of hereditary lues are evidenced in childhood by fibrous arteries and by sclerotic involvement of the myocardium and of the valves—the mitral, the aortic, seldom both in the same case prior to late adult years. I reported several years ago a study of a child of four years dying of a rupture of an aortic aneurysm. The aortic valve was so completely closed by sclerotic thickening and deformity as to cause wonder that enough blood could have passed its ring to maintain life. The child's arteries were of the pipe-stem order. The mother was apparently healthy, but the father had a slight degree of saddle-nose, and developed epileptiform convulsions at thirty-one years. He was in all likelihood a case of latent hereditary syphilis or a none too frank instance of the acquired type.

In a few cases of this nature I have neither been able to obtain an admission from the patient that luetic infection has taken place, nor have I had any reason to think the statement of his freedom from infection was incorrect. I have seen cases that clinically resembled tonsillitis and rheumatism, with endocarditic involvement, which later at the autopsy showed an isolated sclerosis of the mitral valve. I remember one little girl that gave only a history of recurrent tonsillar swelling, yet whose arteries and mitral valve were as rigid as those of an old person.

To the question, What is the exact nature of such a case, I

reply, I do not yet know. Only a searching study of sections of the arteries and the valve leaflets of such cases will exclude the spirochete, and only the Wassermann test, employed as a routine measure in cardiac disease, will assist in the diagnosis *in vivo*.

FIG. 3.—Sclerosis of the mitral valve. Great hypertrophy of the left ventricular wall.

These sclerotic changes occur in the absence of the classical signs of acquired syphilis. Syphilis not infrequently causes a symptom-complex indistinguishable (except by laboratory means) from that of both acute inflammatory rheumatism and of tonsillitis, and it can and does leave in some instances permanent traces in the form of thickening and deformed mitral leaflets. Whether it is the usual cause of these isolated scleroses is another matter. I have also noted two instances of sclerotic involvement of the mitral valve in sisters, members of a family in which a large number of the recent ancestry had been tuberculous. One parent, the mother, had died in early life, probably though not certainly of tuberculosis. The father died of a systemic nervous disease that may easily have been luetic in origin. The children of this parentage, five in number, have all shown evidence of cardiac valvular disease, which in at least two instances was limited to the mitral

valve. Thus we have the possibility of a tuberculous influence and the certainty that syphilis may and sometimes does produce such a picture alone.

Whether other infections may constitute or independently cause isolated valvular sclerosis can only be determined by the studies of many observers of a large series of cases. Dr. James M. Anders informs me that he has studied two interesting cases of this type of mitral involvement in which the causative factor remained unknown. Whether or not acute infectious rheumatism can and ever does cause sclerosis of the valvular tissues is also a point to be considered. As a rule, fibrosis does not form even a prominent feature of the damage consequent upon this disease. Vegetative endocarditis is so generally a rheumatic sequela that this type of valvular disease may almost be regarded as rheumatism of the endocardium. We may therefore pass it over as an unimportant factor in the etiology of the condition under consideration. The text-books have seemingly overlooked mitral sclerosis and implication of the bicuspid valve.

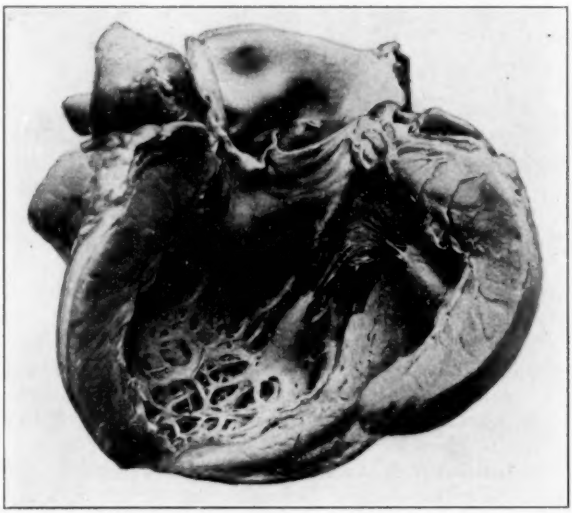


FIG. 4.—Fibrosis of the mitral valve. Marked dilatation of the left ventricle and relative mitral insufficiency. Great hypertrophy of the papillary muscles.

POSSIBILITY OF ANTEMORTEM DIAGNOSIS. From my experience in the cases studied by me I am inclined to believe that all patients, irrespective of age, with sclerosis of the mitral valve, whether associated with other valvular involvements or isolated, will be found also to present sclerotic arteries. The child of four years

already mentioned as dying from a ruptured aortic aneurysm had brachial and radial arteries that would have done credit to an intemperate old man. This early arteriofibrosis, together with a premature overaccentuation of the second aortic sound and a tendency to hypertrophy of the left ventricle, will in the presence of the signs of developing mitral disease afford reason to anticipate fibrosis rather than a deposit of lymph and bacteria as the condition of affairs likely to be revealed at the autopsy. A definite history of syphilis or tuberculosis in the patient or in his or her parents will go far toward rendering presumptive evidence certain.

Preventive Measures and Treatment. The only certain preventive measure would be prevention of marriage among the unfit. The question of the right to marry among those who are morally certain to transmit stigmata, if not actual disease, is not a proper subject for a strictly clinical discussion. It has its bearing, however, immediately we begin to consider the prophylaxis of valvular disease.

I doubt whether in the presence of sclerotic arteries in child or adult much can be done in the way of forestalling sclerotic heart change. Both the cardiac muscle and the valve leaflets seem destined to proceed in the course which they have begun before the presence of a toxic factor has been recognized. It is probably too late for us to expect much valvular benefit from antiluetic medication, though in the presence of repeatedly positive Wassermann reactions, thorough courses of arsenic and mercury should be instituted. The studies of Finger and other reliable investigators would seem to indicate that hereditary syphilis is an incurable disease. In the absence of such symptoms as those of lues, we are peculiarly helpless against the insidious march of an irremediable affection. The treatment therefore again reverts in the present very imperfect state of our knowledge of the etiology of these conditions to a new interest in human eugenics, and to precautionary breeding measures that will prove as intelligent in the reproduction of the human species as in the raising of healthy cattle, horses, and hogs.

REVIEWS

PROGRESSIVE MEDICINE. A QUARTERLY DIGEST OF ADVANCES, DISCOVERIES AND IMPROVEMENTS IN THE MEDICAL AND SURGICAL SCIENCES. Edited by HOBART AMORY HARE, Professor of Therapeutics and Diagnosis in the Jefferson Medical College of Philadelphia; Physician to the Jefferson Medical College Hospital; assisted by LEIGHTON F. APPLEMAN, M.D., Instructor in Therapeutics, Jefferson Medical College, Philadelphia, etc. Vol. I, March, 1913; pp. 353. Philadelphia and New York: Lea & Febiger.

THE first volume of Progressive Medicine for this year opens with an article of 106 pages on the surgery of the head, neck, and thorax by Charles H. Frazier. The hypophysis, meningitis, and trigeminal neuralgia are among the most important subjects considered under the head. A wealth of interesting material is reviewed in the course of his discussion of the thorax, particularly surgery of the heart and great vessels, intratracheal insufflation, and surgery of chronic disease of the lungs. The most noteworthy topics discussed in the remainder of his contribution are cancer of the mouth, lips, tongue, and pharynx, cervical lymphadenitis, cervical ribs, goitre, and breast tumors.

John Ruhräh has contributed a most interesting article of 112 pages on infectious diseases. He lays particular stress upon cerebrospinal fever, diphtheria, kala-azar, leprosy, pneumonia, poliomyelitis, scarlet fever, tetanus, typhoid fever, tuberculosis, and pertussis. No one can read this summary without being impressed by the notable advances that are being made in this branch of medicine.

Diseases of children are considered by Floyd M. Crandall in a short article. After dwelling upon infant mortality and child welfare, he takes up among other subjects, hemorrhage in the newborn, edema in infants, chorea, achondroplasia, the exudative diathesis, and infant foods.

Rhinology and laryngology are ably discussed by George B. Wood. He first reviews the question of the choice of anesthetics for nose and throat operations; then among other interesting topics, he considers submucous resection of the nasal septum, nasal diphtheria, various forms of rhinitis and accessory sinus disease; septic

sore throat and Vincent's angina are among the subjects taken up under the pharynx. He then discusses the tonsils and their surgery, and finally devotes considerable attention to the larynx.

A contribution on otology, by A. B. Duel, completes the volume. He fully enters into the subjects of meningitis, the labyrinth, the treatment of specific disease of the ear with salvarsan, otosclerosis, and neoplasms of the ear.

The volume is one of decided value and the authors are to be commended for their careful selection of so many subjects of great present-day interest. G. M. P.

APPENDICITIS. By JOHN B. DEAVER, M.D., Sc.D., LL.D., Professor of the Practice of Surgery, University of Pennsylvania. Fourth edition; pp. 379; 14 illustrations. Philadelphia: P. Blakiston's Son & Co., 1913.

SEVENTEEN years ago the first edition of this book was criticised—on the whole unfavorably—by the present writer. The specific counts of the indictment against the book were chiefly as follows: (*a*) A lack of information as to the proportion of recoveries from genuine attacks, without operation; as to the number of such cases who have second attacks; as to the proportionate fatality of such recurrences as compared with primary outbreaks; as to the relative advantages of immediate operation and of delay in cases seen some days after the primary seizure, and which show signs of the formation of a localized abscess; as to the existence or non-existence of "stercoral typhlitis," and of the "simple catarrhal" or "mechanical" forms of appendicitis, those thought to be caused by a stretching or a twisting of the meso-appendix; (*b*) insufficient description of the relapsing and recurrent forms of the disease and of its complications; (*c*) a too free use of other authors without adequate acknowledgment; and (*d*) a too absolute insistence upon the rule that "where practicable all cases of appendicitis should be operated upon as soon as the diagnosis has been established," though as to the last point, the reviewer added: "This may be good surgery; time will show."

It has been most interesting to read the present edition of this book with these criticisms of nearly two decades ago in mind. It is fair to say:

First, that such retrospective comparison demonstrates that, as has been the case with many reviewers, from the days of "The Dunciad" or of "English Bards and Scotch Reviewers" down to this relatively insignificant instance, it is apparent that it is easier for a reviewer to be, or to seem, clever than to be just. There

were faults to be condemned, of course, but there were also excellences to be commended, and it is now apparent that the latter were not given their proper proportionate prominence.

Second, whatever may have been the genuine faults of the edition of 1896, they have in the changes and additions of the intervening years practically disappeared from the edition of 1913.

The experience and the resulting statistics, not only of the author, but of the whole profession, have so vastly broadened during this period that questions which were then pressing for solution are no longer debatable, and analytical studies of series of individual cases, then much to be desired would now be a waste of time and effort.

The increased experience of the author is shown in the fact that whereas the first edition was based on a "series of 500 cases" his present views have back of them "an experience involving about 10,000 cases of appendicitis" (p. 224).

As to the supposed individual faults of the early edition, we now find by contrast a thorough and entirely satisfactory description of all the varieties of appendicitis and of its complications, clear and succinct, but comprehensive.

Familiarity with the literature of the subject is shown, but the fullest possible credit is given where credit is due. The teachings as to operation are no less positive, but, as to this, it should certainly be admitted that the surgeon who has seen 10,000 cases of a particular condition has at least twenty times the right to be uncompromising or even dogmatic in his advice to practitioners and students that he had when he had seen only 500 cases. In reality, his right to be positive increases far more than by that simple arithemtical proportion. It is certain that when, as in these instances, he speaks clearly and unequivocally, and bases his teachings not only on his own experience, but on well-sustained and well-defined clinical and pathological theories, the professional world must listen respectfully.

Some specific comparisons with the earliest edition may be made with advantage.

The section on Differential Diagnosis contains more than twice the number of words and a vastly more useful comparison of the various conditions that may be confused with appendicitis. Clinical Etiology is similarly enlarged and improved. The section on Pathology, increased more than four-fold, is a masterly exposition of our knowledge of this underlying subject. The summaries of the Principles of Symptomatology (p. 195) and of Diagnosis (p. 216), the Recapitulation of the Pathological Factors (p. 170), the discussion of the Medical Treatment of Appendicitis (p. 355), may be singled out for approval as special examples of the admirable handling of the subject that characterizes the book as a whole.

Particular mention should also be made of the section on Peritonitis, which is exceedingly interesting and instructive. Most surgeons of today will agree with the author when he states (p. 32) that saline enteroclysis by Murphy's continuous method is the greatest advance in the postoperative treatment of abdominal conditions in the last decade. He says that by its use he has been able to reduce his mortality in cases of diffuse peritonitis to less than 2 per cent.—a truly noteworthy showing.

The most radical change of opinion the reviewer has noticed relates to the early administration of laxatives, and it is instructive to note that in 1896 that was a question as to which he and the author were in absolute accord.

In the first edition, Dr. Deaver said: "I am perfectly familiar with the unfavorable opinions of a number of other writers upon the advisability of the administration of laxatives in appendicitis, but my experience has taught me that it forms the only successful, and therefore justifiable, treatment when operation cannot be performed. I do not hesitate, therefore, to offer it to my readers as sound and rational therapeutics. I repeat that laxatives should be given early, and in sufficient quantity to produce thorough evacuation of the bowel, for they accomplish the most good when given thus. . . . The benefit of unloading the bowel far outweighs the danger of breaking up any adhesions that may be forming."

In the present edition he says: "The most important points to be observed in the suspected presence of acute appendicitis are negative rather than positive, and consist in the prohibition of everything by mouth, including water, and especially the avoidance of all purgative medicine."

He also prohibits anodynes, and at present the only difference in our respective views would be that at the end of the above-quoted sentence we would say "especially the avoidance of all anodynes," leaving to purgatives a secondary place as to possible harmfulness. At any rate, this example shows what is apparent throughout the book, that the author has the courage of his convictions and is still young enough to change his mind.

We have noticed but few typographical or other errors. On page 40 the reference to the period when prompt "ablation of the diseased organ meant cure without hope of relapse" should probably read "*fear* of relapse." The latter was scarcely to be "hoped" for by any one. On page 217, the statement that fever is "next to never" the first symptom, sounds a trifle colloquial. On the same page we would prefer to see "excrutiating" spelled "excruciating," though in these days of changing orthography, that is of slight, importance. On page 357, "Purgatives . . . hastens," should, of course, take the singular form of the verb. But the proof-reading in the main has been well done and the whole book is

creditable as to its style and manner as well as to its teachings. Indeed, as to the latter, it is not over-praise to say that it is at once the most clear and compact and the most authoritative presentation of its important subject that is today before the medical profession. J. W. W.

AUGUSTUS CHARLES BERNAYS. A MEMOIR. By THEKLA BERNAYS. Pp. 309. St. Louis: C. V. Mosby Company, 1912.

THIS memoir of the late Dr. Bernays written, by his devoted sister, constant companion, and chief confidant, has been read with a great deal of interest by the reviewer. The early life of the subject of these pages, treated in the first six chapters, of which chapters there is a brilliant interlude by Prof. Furbinger, were read with the most pleasure. They seemed to be, in a sense, an extremely intimate and therefore affectionate portrayal of the early days of any man, wherein the vagaries of youth and the budding of maturity are all set forth in a way that reminds us of certain pages of "Jean Christophe." We were perplexed to discover what might be the motive of the authoress in offering to the public and to the cold gaze of the reviewer, this scene of family with its manners, trials, successes, affections and love, for such things are generally beheld only by a favored few. This perplexity was unfortunately increased by our lack of familiarity with the work, achievements, or even name of Augustus Charles Bernays. The bibliography appended to the memoir shows the wide scope of his medical writings, and but added to the chagrin of the reviewer in being so lamentably unfamiliar with "one of the most brilliant surgeons that this country has ever produced," to quote from the publisher's letter.

We do not wish to hurt the feelings of a sister who can so lovingly and appealingly record the virtues of an adored brother, and who can so tenderly and with such a gentle way of offering excuse for what she indeed thinks to be the opposite, pen his peccadillos. Miss Bernays' quaint remark on things medical, her naïve comparison between gastrostomy and appendectomy, and her ingenuous chapter on Dr. Bernays' Views on Fever, wherein, unconsciously, she sets forth her own at great length, all these add to the charm if not to the value of the volume. We have gathered the impression, we who have perused carefully, calmly, and dispassionately these memoirs, that they are to be considered as a protest against what the authoress regards as harsh treatment by the medical profession of her brother during his life. The reviewer being unfamiliar with Dr. Bernays' medical activities in St. Louis cannot judge of this treatment, but he believes *De mortuis nil*

nisi bonum. Perhaps the friends, enemies, colleagues, and students of Dr. Bernays will find the book of interest, but to the general medical public it cannot make a lasting appeal unless it does so as the literary product of a mourning sister still crying as she does in her pages, "Never was there another such a brother."

E. H. G.

THE PATHOLOGY OF THE LIVING, AND OTHER ESSAYS. By B. G. A. MOYNIHAN, M.S. (London), F.R.C.S., Honorary Surgeon to Leeds General Infirmary; Professor of Clinical Surgery at the University of Leeds, England. Pp. 260. Philadelphia and London: W. B. Saunders Company.

THIS little volume consists of nine now familiar essays by one of the most noted abdominal surgeons of the world, and should prove a welcome addition to many a medical library. The essays were for the most part originally delivered as addresses before various medical societies, and although dealing with surgical subjects, are of no less interest to the general practitioner than to the surgeon. On the contrary, it is to the general practitioner that they should be recommended particularly, for it is safe to say that if the lessons taught in the essay on Pathology of the Living were firmly fixed in the minds of more general practitioners, more gastric carcinomas would come to the operating table at a period when they are operable, and fewer cases of gastric ulcer would be treated for months as chronic indigestion.

The essays which comprise this book are so well known to the profession at large that they require no detailed comment. It is interesting to note, however, that many of the statements made for the first time by Moynihan in these addresses, all of which were delivered prior to the latter part of 1908, have been amply confirmed by the subsequent investigations not only of Moynihan, but also of a large group of abdominal surgeons in both Europe and America.

G. M. P.

SURGICAL OPERATIONS WITH LOCAL ANESTHESIA. By ARTHUR E. HERTZLER, M.D., Surgeon to the Halstead Hospital and to the Swedish Hospital, Kansas City. Pp. 205; 104 illustrations. New York: Surgery Publishing Co., 1912.

THIS work has been undertaken to supply a demand for a systematic, concise description of the uses and possibilities of local anesthesia. The author has made it a one-man book in that he

creditable as to its style and manner as well as to its teachings. Indeed, as to the latter, it is not over-praise to say that it is at once the most clear and compact and the most authoritative presentation of its important subject that is today before the medical profession. J. W. W.

AUGUSTUS CHARLES BERNAYS. A MEMOIR. By THEKLA BERNAYS. Pp. 309. St. Louis: C. V. Mosby Company, 1912.

THIS memoir of the late Dr. Bernays written, by his devoted sister, constant companion, and chief confidant, has been read with a great deal of interest by the reviewer. The early life of the subject of these pages, treated in the first six chapters, of which chapters there is a brilliant interlude by Prof. Furbinger, were read with the most pleasure. They seemed to be, in a sense, an extremely intimate and therefore affectionate portrayal of the early days of any man, wherein the vagaries of youth and the budding of maturity are all set forth in a way that reminds us of certain pages of "Jean Christophe." We were perplexed to discover what might be the motive of the authoress in offering to the public and to the cold gaze of the reviewer, this scene of family with its manners, trials, successes, affections and love, for such things are generally beheld only by a favored few. This perplexity was unfortunately increased by our lack of familiarity with the work, achievements, or even name of Augustus Charles Bernays. The bibliography appended to the memoir shows the wide scope of his medical writings, and but added to the chagrin of the reviewer in being so lamentably unfamiliar with "one of the most brilliant surgeons that this country has ever produced," to quote from the publisher's letter.

We do not wish to hurt the feelings of a sister who can so lovingly and appealingly record the virtues of an adored brother, and who can so tenderly and with such a gentle way of offering excuse for what she indeed thinks to be the opposite, pen his peccadillos. Miss Bernays' quaint remark on things medical, her naïve comparison between gastrostomy and appendectomy, and her ingenuous chapter on Dr. Bernays' Views on Fever, wherein, unconsciously, she sets forth her own at great length, all these add to the charm if not to the value of the volume. We have gathered the impression, we who have perused carefully, calmly, and dispassionately these memoirs, that they are to be considered as a protest against what the authoress regards as harsh treatment by the medical profession of her brother during his life. The reviewer being unfamiliar with Dr. Bernays' medical activities in St. Louis cannot judge of this treatment, but he believes *De mortuis nil*

nisi bonum. Perhaps the friends, enemies, colleagues, and students of Dr. Bernays will find the book of interest, but to the general medical public it cannot make a lasting appeal unless it does so as the literary product of a mourning sister still crying as she does in her pages, "Never was there another such a brother."

E. H. G.

THE PATHOLOGY OF THE LIVING, AND OTHER ESSAYS. By B. G. A. MOYNIHAN, M.S. (London), F.R.C.S., Honorary Surgeon to Leeds General Infirmary; Professor of Clinical Surgery at the University of Leeds, England. Pp. 260. Philadelphia and London: W. B. Saunders Company.

THIS little volume consists of nine now familiar essays by one of the most noted abdominal surgeons of the world, and should prove a welcome addition to many a medical library. The essays were for the most part originally delivered as addresses before various medical societies, and although dealing with surgical subjects, are of no less interest to the general practitioner than to the surgeon. On the contrary, it is to the general practitioner that they should be recommended particularly, for it is safe to say that if the lessons taught in the essay on Pathology of the Living were firmly fixed in the minds of more general practitioners, more gastric carcinomas would come to the operating table at a period when they are operable, and fewer cases of gastric ulcer would be treated for months as chronic indigestion.

The essays which comprise this book are so well known to the profession at large that they require no detailed comment. It is interesting to note, however, that many of the statements made for the first time by Moynihan in these addresses, all of which were delivered prior to the latter part of 1908, have been amply confirmed by the subsequent investigations not only of Moynihan, but also of a large group of abdominal surgeons in both Europe and America.

G. M. P.

SURGICAL OPERATIONS WITH LOCAL ANESTHESIA. By ARTHUR E. HERTZLER, M.D., Surgeon to the Halstead Hospital and to the Swedish Hospital, Kansas City. Pp. 205; 104 illustrations. New York: Surgery Publishing Co., 1912.

THIS work has been undertaken to supply a demand for a systematic, concise description of the uses and possibilities of local anesthesia. The author has made it a one-man book in that he

gives merely his own technique and ideas. He describes only those operations which he thinks are applicable to local anesthesia.

In discussing the advantages of local over general anesthesia he fails to appreciate the advantages of nitrous oxide and oxygen as a general anesthetic.

The technique is described in minute detail, even including sterilization of skin and the demeanor of the operator. Of the various drugs employed the quinine urea solution is rightly regarded the safest. Cocaine is advised for the operations demanding only small amounts of the anesthetic. Novocaine and eucaine are much favored because of their non-toxicity.

The remaining thirteen of the seventeen chapters are concerned with the description of various operations. Each operation is described fully and individually. The anatomy, neural especially, is outlined, the drugs, amounts used, and technique described step by step in detail. Thus the operator can read up his entire procedure in the one work.

The style and diction of the work are good and make pleasant reading. However, the book has been poorly edited, as typographical errors are noticeable, such as misspelled words and the transposition of parts of sentences.

The illustrations fall short of accuracy in one or two particulars, and consequently would tend to confuse rather than aid the reader.

However, the work is an instructive one in many respects, and one that every surgeon should have at his command.

E. L. E.

ON THE PHYSIOLOGY OF THE SEMICIRCULAR CANALS AND THEIR RELATION TO SEA SICKNESS. By JOSEPH BYRNE, A.M., M.D., LL.B. Pp. 569. New York: J. I. Dougherty, 1912.

As stated in the preface to this volume, its author undertook to write an article on the etiology of seasickness some years ago, but "not satisfied with a mere expression of his view without appeal to experimental fact, and believing that the semicircular canals were in some way involved in the causation of the malady, he undertook a series of experiments, using rotations, aural irrigations, stimulation of the retina by strong light, galvanism applied to the mastoid areas, etc., to determine whether by such means phenomena resembling those of seasickness could be experimentally reproduced."

The text of this elaborate work is divided into three sections: (I) General anatomical and physiological considerations; (II) physiology of the semicircular canals; (III) seasickness. Pp. 339 to 525.

We may dismiss the first part without other comment than it is fully lucid and up to date.

The second part, physiology of the semicircular canals, commences with a summary of the most authoritative results of experimentation on animals, and then details a long series of personal experiments upon human subjects, the results of which are minutely described, as they were manifested in organs near and remote from the aural structures. Numbers of these are tabulated in series of elaborate protocols, any summary of which here would be too superficial for usefulness.

The third part is an equally elaborate experimental study of seasickness and its results on board ship. The general conclusions convinced the author that the effects of real seasickness are quite analogous to the artificial sickness caused by rotation, aural irrigations, and by galvanism applied over the mastoid areas.

While all this work is learned, laborious, and interesting, it is doubtful whether this physical cause of seasickness is the real one.

To conclude, the treatment suggested for seasickness is quite indeterminate and unsatisfactory.

In maritime circles, however, seasickness of landsmen is often immediately cured by the psychic effects of a rope's end or a knock-down blow with the fist. J. S. C.

THE SURGICAL TREATMENT OF LOCOMOTOR ATAXIA. By L. N. DENSLOW, M.D., Late Professor Genito-urinary Surgery and Venereal Diseases, St. Paul Medical College, Minnesota. Pp. 118. London: Baillière, Tindall & Cox, 1912.

THE purpose of this small volume is to present a new theory as to the cause of locomotor ataxia and a new method of treatment based upon this theory. The theory is as follows: "The dystrophic changes that occur in the neurons of the posterior roots and their connections in tabes are the result of continuous sensory impulses conveyed from some peripheral point to the sensory roots in the cord; that such continuous impulses, kept up, perhaps, for years, exhaust the central nerve substance which having no rest or intermission from such impulses, and having no opportunity for recuperation, finally succumbs." The "peripheral point" from which the "sensory impulses" originate, in the great majority of cases, is an inflamed or constricted portion of the urethra. About half of the book is given up to a discussion of the theory and the other half to a citation of cases treated by the author. The results obtained are at least suggestive but are not conclusive. The greatest need in connection with the theory and treatment is confirmation by other workers in this field.

T. T. T.

HANDBOOK OF MENTAL EXAMINATION METHODS. By SHEPHERD IVORY FRANZ, PH.D., Scientific Director and Psychologist, Government Hospital for the Insane; Professor of Physiology, George Washington University. Pp. 165; 33 illustrations. New York: The Journal of Nervous and Mental Disease Publishing Co., 1912.

THIS small book is one of the Monograph Series published by the *Journal of Nervous and Mental Disease*. The journal deserves a deal of credit for the excellence of its selection, for the series includes translations of such works as those of Sigmund Freud, Plaut, Jung, and others.

The present volume, which is No. 10 of the series, is a manual of the modern methods of examination of mental diseases. It is excellently done, and is a safe guide for those who intend to study psychiatry along modern scientific methods. T. H. W.

THE THERAPY OF SYPHILIS. By PAUL MULZER, M.D., of Berlin. Translated by A. NEWBOLD.

THE *Therapy of Syphilis*, by Dr. Paul Mulzer, consists of a brief essay of 44 pages on the mercurial and the more important arsenical forms of antisyphilitic treatment, other than salvarsan, with a history of their development, shortcomings, and discard; and a rather lenghty treatise of 160 pages on the therapy of syphilis by the Erhlich-Hata arsenical preparation, dioxydiamidoarsenobenzol or "606."

Mulzer gives a short history of the discovery of "606," and animal experimentation by Ehrlich and Hata, a description of the various methods of preparation of the drug for the different forms of injection, and clinical conclusions on the value, contraindications, criticisms, and results of its use, based on one year's observations (January, 1910 to December, 1910), of many well-known European authorities.

The rapid progress in our knowldege of the handling of this drug, both in the technique of its administration and the clinical discretion in its use, since December 1910, which Mulzer must have foreseen, reflects only credit to his industry in the production of such a work.

This book should be, at least, of historic value and interest to the profession. J. L. L.

PROGRESS

OF

MEDICAL SCIENCE

MEDICINE

UNDER THE CHARGE OF

W. S. THAYER, M.D.,

PROFESSOR OF CLINICAL MEDICINE, JOHNS HOPKINS UNIVERSITY, BALTIMORE, MARYLAND,

AND

ROGER S. MORRIS, M.D.,

CHIEF OF THE DEPARTMENT OF INTERNAL MEDICINE, CLIFTON SPRINGS SANATORIUM AND HOSPITAL, CLIFTON SPRINGS, NEW YORK.

Alimentary Galactosuria in Experimental Phosphorus Poisoning.—R. ROUBITSCHEK (*Deutsch. Archiv f. klin. Med.*, 1912, cviii, 225), at the suggestion of Reiss, has studied the galactose tolerance of dogs during phosphorus poisoning. He found that galactosuria occurred with the acute degenerative changes produced in the liver cells. This fact, taken with the results of the studies of Reiss and Jehn, leads Roubitschek to believe that it is only the acute conditions which injure the liver parenchyma diffusely that interfere with the power of the organ to synthesize galactose to glycogen. In chronic conditions such as cirrhosis, chronic passive congestion, etc., there is regeneration of liver cells which may compensate functionally to a sufficient degree to prevent the occurrence of galactosuria.

The Bradycardia of Lead Colic.—It is not uncommon to observe, in the course of lead colic, an outspoken bradycardia. Tanquerel, in an analysis of 1179 cases, has found a pulse of 20 to 60 in 678 instances, 65 to 70 in 376, and 80 to 100 in 125. LION and MARCORELLES (*La Presse Méd.*, 1913, Nr. 12, 109) have applied graphic methods and the atropine test to 4 cases. The tracings proved the condition a true bradycardia, with a normal phlebogram and no auriculoventricular disassociation. Each ventricular systole was preceded by a single auricular systole. The heart was normal but slow. Arterial hypertension and the bradycardia were independent. Both are probably manifestations of saturnine intoxication. In each case, atropine produced a tachycardia, the pulse changing from 46 to 52 to 75 to 120. This leads them to conclude the toxic impregnation is either of the vagus trunk, or the nerve endings in the node of Keith and Flack.

Acetone and Diacetic Acid.—Bonnamour and Joubert (*La Presse Med.*, 1913, Nr. 14, 130) have modified the Legal and Gerhardt test for acetone bodies in the urine. They emphasize that the two are distinct, the first indicating the presence only of acetone, the second diacetic acid. The Legal reagent as modified was made with 10 grams of glacial acetic acid and 10 c.c. of 10 per cent. sodium nitroprusside. In a colored bottle, this can be kept several months. To 15 c.c. of filtered urine are added 20 drops of reagent. After mixture, ammonia is floated upon the surface drop by drop. The presence of acetone even in dilution of 1 to 2000 causes a violet disk at the line of separation of the two. With the test acetone was detected in the urine of numerous non-diabetic patients. They concluded that acetonuria may be the index of acidosis, but in itself is not an indication of imminent diabetic coma. It is not evidence of the presence of diacetic acid. Gerhardt's test for diacetic acid was performed by adding to urine diluted with four volumes of water a 10 per cent. solution of perchloride of iron drop by drop. Normal urine, containing even acetone, gives a white cloud. In urine with a trace of diacetic acid, the precipitate is a definite black violet cloud. The reaction is much more delicate than the original Gerhardt test, and is given only by diacetic acid. The two reactions are distinct for each acetone body.

Syphilitic Aortitis.—Longcope (*Arch. Int. Med.*, 1913, xi, 15) has observed no definite beneficial effect from salvarsan in 20 cases of syphilitic aortitis. Neither the cardiac competency nor the anatomical condition have been changed. Signs of aortic insufficiency have not been reduced and aneurysms have not grown smaller. The most striking result has been upon the pain and attacks of paroxysmal dyspnea. The relief has been immediate and tends to be permanent. The persistence of a positive Wassermann reaction or the rapid return of a positive reaction after a temporary abatement during treatment have impressed the author. The improvement in symptoms manifests itself very rapidly, usually within four to six days, but this is associated with an increase in the severity of symptoms within twenty-four hours to forty-eight hours after the intravenous injection. This is particularly true of angina pectoris and paroxysmal dyspnea. The rapid amelioration of the pains, attacks of angina pectoris, and paroxysmal dyspnea suggests certain explanations for these symptoms. Autopsies have not been convincing of any direct association between diseases of the coronary arteries and these attacks. The suggestion is strong that the symptoms are associated with the inflammatory reaction at the root of the aorta and directly dependent upon it. The increase in the severity of symptoms after injection resembles a Herxheimer reaction, the increase in symptoms due to increased reaction of the tissues toward the liberation of toxins in excess from rapidly destroyed spirochetes. The dyspnea caused by bronchospasm, and the contraction of the peripheral arteries producing heightened blood pressure from aortic irritation in experimental animals is a close reproduction of the paroxysmal dyspnea, as it occurs in syphilitic aortitis. Longcope believes these symptoms dependent upon a reflex generated at the root of the aorta by the syphilitic inflammatory process. At any rate, the true etiology probably lies in the diseased aorta, and this

is supported by the Herxheimer reaction, combined with the rapid improvement and later recurrences. Longcope in conclusion considers that syphilis produces a characteristic lesion of the aorta which is responsible for most aneurysms, about 75 per cent. of cases of aortic insufficiency in adults, many cases of dilatation of the aorta, and a certain group of cases of angina pectoris. The infection of the aorta probably taken place during the secondary stage, and the process usually remains latent, or unrecognized for an average of sixteen or seventeen years. Thus syphilitic aortitis is probably a common cause for a positive Wassermann reaction in so-called latent syphilis.

Congenital Family Steatorrhea.—GARROD and HURTLEY (*Quarterly Jour. Med.*, 1913, vi, 242) report the case of a boy, aged eight years, subject from infancy to true steatorrhea, the passage from the bowel of liquid fat which solidifies on cooling. His parents are first cousins. A brother was similarly affected from birth. The boy is well nourished and exhibits no other morbid conditions. There are no signs of pancreatic disease besides the steatorrhea. On a diet containing very little fat his stools assume a normal appearance and consistency. Analyses show he excretes in his stools 25 per cent. of the fat taken by mouth. Improved saponification is not followed by improved absorption. The boy is presumably the subject of a rare inborn error of fat absorption, probably a Mendelian recessive characteristic. Investigation did not reveal where the error lay.

On Uric Acid Excretion in Hypophyseal Disease.—W. FALTA and J. NOWACZYNSKI (*Berlin. klin. Woch.*, 1912, xl, 1781) find no data on uric acid excretion in hypophyseal disease. Their own findings on a limited material are of considerable interest. The patients were placed on a purin-free diet, and the uric acid of the urine was determined by the method of Hopkins-Folin-Shaffer. In 3 cases of acromegaly they found the endogenous uric acid to be twice the normal average or greater. In 1 patient there was an endonasal resection of part of the hypophysis. No clinical evidence of improvement was noted shortly after the operation, nor was there any decrease of the uric acid output, compared with the pre-operative periods. In this patient there was a prompt increase in uric acid following the administration of 20 grams of sodium nucleinate. In 2 cases of dystrophia adiposogenitalis associated with hypophyseal tumor (one hypophysis cystic at autopsy, the other "a tumor in the hypophysis region"), they found, on the other hand, that the endogenous uric acid was normal or subnormal, with only a slight response to the administration of sodium nucleinate. They assume that the acromegalics are suffering from an increased functional activity of the hypophysis, and on this basis they suggest that the determination of endogenous uric acid may be of some diagnostic value, provided their results are borne out by the study of a larger material.

Autoserotherapy and Absorption of Ascites.—VITRY and SÉZARY (*Revue de Médecine*, 1913, xxxiii, 86) studied the absorption of ascitic fluid in a case of cirrhosis of the liver. The patient, a woman, aged fifty-two years, had noticed the insidious swelling of the abdomen

for six months. The abdomen was bulging with a large ascites, estimated at 8 to 10 liters. There was a prominent collateral circulation. The cause of the hepatic lesion was obscure, probably alcoholic. On a milk diet, the urinary output was 1000 to 1400 c.c. Subcutaneous injections of ascitic fluid were given; 10 c.c. were injected in the abdominal wall immediately after withdrawal. This was done every two days. On the third day an abundant diuresis began and increased to the point when eleven days later the therapy could not be continued for lack of ascitic fluid. The body weight diminished in a corresponding way. An increased chloride content coincided with the polyuria and loss of weight. The acidity of the urine was diminished by the elimination of the alkaline ascitic fluid. The authors were impressed with the favorable influence, although they claimed no effect upon the condition of the liver. However, the disappearance of evidence of portal obstruction was maintained, even when the patient returned to a salt-containing diet.

On the Presence of Typhoid Bacilli in the Mouth of Typhoid Fever Patients and Typhoid Convalescents.—B. PURJESZ and O. PERL (*Wien. klin. Woch.*, 1912, xxv, 1494) have made cultures from the gums, tonsils, and tongue of patients suffering with typhoid fever. Sterile swabs were rubbed on the mucosa, and then, tubes containing Conradi-Drigalski medium were inoculated. The organisms were finally identified by agglutination tests with the serum of a rabbit inoculated with typhoid bacilli, whose serum agglutinated in a dilution of 1 to 3000 in twenty minutes. They examined 17 cases in all. In about 50 per cent. of the cases a positive result was obtained. During the febrile period a positive culture was obtained as late as the twentieth day. The authors have obtained the organisms from the mouth as late as the fourth to eighth week of convalescence, confirming the observations of Gould and Quales, which appeared during the course of the authors' study. The findings are important both from the standpoint of diagnosis and epidemiology.

Blood Findings in Adiposity.—CARO (*Berlin. klin. Woch.*, 1912, xl, 1881) has made counts and histological studies of the blood in 34 patients suffering with adiposity. There were 19 cases of "constitutional" adiposity, 6 cases following the climacteric, and 9 cases of "alimentary" adiposity. The blood showed constantly a lymphocytosis, as high as 62 per cent., with a decreased percentage of polynuclear neutrophile cells. The total number of leukocytes tended to be high—up to 12,000. The red cell count was frequently slightly reduced. Except for lymphocytosis in 1 case of hypophyseal adiposity and in 2 adipose eunuchs, the author has been unable to find reports of blood examinations in adiposity in the literature. Under the influence of thyroid tablets, the lymphocytosis observed by him decreased or disappeared, and there was a corresponding increase of the neutrophiles, in a few cases in which the gland was administered. Kocher has observed similar blood changes in myxedema after administering thyroid gland—for here, too, in untreated cases there is a lymphocytosis.

SURGERY

UNDER THE CHARGE OF

J. WILLIAM WHITE, M.D.,

FORMERLY JOHN RHEA BARTON PROFESSOR OF SURGERY IN THE UNIVERSITY OF PENNSYLVANIA AND SURGEON TO THE UNIVERSITY HOSPITAL,

AND

T. TURNER THOMAS, M.D.,

ASSOCIATE PROFESSOR OF APPLIED ANATOMY IN THE UNIVERSITY OF PENNSYLVANIA; SURGEON TO THE PHILADELPHIA GENERAL HOSPITAL AND ASSISTANT SURGEON TO THE UNIVERSITY HOSPITAL.

A Procedure for Wide Extirpation of Cancer of the Prostate.—GAYET, CHAMPEL, and FAYOL (*Jour. d'Urolog.*, 1913, iii, 333) say that up to the present time three perineal paths have been employed for the removal of a cancerous prostate. These they designate as the anterior, middle, and posterior. The anterior is through the anterior triangle of the perineum. In the middle, which is more frequently employed, a transverse incision is made between the two tuberosities of the ischium. In the posterior the incision is made from the tuberosity of the ischium to the coccyx. Gayet, Champel, and Fayol utilize the whole length of the perineum, the incision passing well to the left side of the anus. The following is a brief outline of the operation which they describe: The patient is placed in the lateroventral decubitus or the pelvis may be extremely elevated so that the perineum looks toward the ceiling. The incision which passes from the root of the penis to the coccyx is deepened and the rectum separated and pushed posteriorly. The posterior surface of the prostate, the seminal vesicles and the posterior inferior surface of the bladder are then exposed. If the lateroventral position has been employed, this is now changed to the dorsal with extreme elevation of the pelvis. The perineal floor is now divided in its whole length and the rectum is pushed further backward and to the right side. The finger recognizes the apex of the prostate and membranous urethra which is easy if a sound or catheter has been placed in the urethra. The urethra is opened in front of the apex of the prostate and through this opening an elbowed retractor is introduced. The urethra is divided transversely in its whole circumference and the prostate is separated from the surrounding structures by scissors, the hemorrhage being controlled by a gauze tampon. If the prostate can be depressed far enough, a puncture is made by a bistoury through the anterior wall of the bladder above the portion invaded by the tumor. This opening is enlarged on both sides by scissors, giving a wide opening into the bladder. The posterior portion of the bladder in the region of the trigone and ureters is now well exposed. With catheterization of the ureters, the rest of the separation of the prostate and tumor can be completed under the guidance of the eyes. The separation of the divided ends of the urethra and bladder is considerable, but their elasticity aided by a

moderate liberation of the urethra permits them to be brought together by sutures. But it is not necessary to suture them together in their whole circumference, a large opening in the bladder being left posteriorly. Through this opening a large tube is passed and fixed in position with a catgut suture. The muscles of the perineum are then restored very incompletely and a large drain is introduced penetrating to the side of the vesical tube. The skin margins are then brought together, leaving a large median opening for gauze drainage. The results of this operation in two cases were benign. At the end of five weeks in the first patient all urine passed in the natural way and in the second patient more quickly, without the necessity of catheterization or dilatation.

Traumatic Epilepsy after Head Injuries in the Japanese-Russian War.—Eguchi (*Deutsch. Zeitschr. f. Chir.*, 1913, cxxi, 199) says that head injuries frequently produce traumatic epilepsy. From 1.3 to 4.7 per cent., on the average 3 per cent., of all skull injuries admitted to the reserve hospitals of Japan during the war were followed by traumatic epilepsy. In these, injuries of the bone and dura mater produced irritation of the brain. Wounds of the soft tissues alone gave no cases of epilepsy. The symptoms followed not only injuries of the cortex in the motor area, but many times injuries in other areas. The traumatic epilepsy appears usually after cicatricial tissue develops, Its occurrence at an earlier stage is very rare. It was more frequent after sagittal than after transverse gun-shot wounds. The frequency was in proportion to the size of the brain injury. The autopsy did not show in any case any changes in the medulla, pons, or cerebellum. Scars of the head are very irritable in cases of traumatic epilepsy, and are very tender on palpation. If a bone defect is covered only with a flap of soft tissues, the pulsation of the brain can be seen. The soft tissue flap used corresponded to that of Kocher. Adhesions between the dura or skin and the brain frequently produce epilepsy, as does traumatic new formation, such as thickening of the inner table of the skull, cysts, abscesses, etc. Foreign bodies, pieces of bone, and in war especially, splinters of bone or pieces of missiles, cause epilepsy by pressure on the dura and brain. All of Eguchi's cases developed a nervous disposition. Epilepsy develops easily after head injuries in nervous people. The shortest period from the day of the injury to the beginning of the epilepsy was 3 days, the longest was 442 days, the average 161 days. Medicines, especially the bromides, cannot suppress the attacks before operation, but after operation has been done the attacks, which recur in some cases, can be more quickly controlled by the bromides. Operation gave good results in Eguchi's cases. A cure was obtained in 11 cases, or 90.9 per cent., and no results in 1 case, or 9.1 per cent. The death in this last case was not the result of the operation, but of the bursting of an aneurysm. In all of the other cases no attack had occurred two years after operation. One should operate on all cases of traumatic epilepsy from head injuries in which he detects scars, or wounds, or other changes. If the epilepsy developed a short time before the operation, a quick result should be expected. If the attacks have been occurring for a much longer period, the operation may be followed by several attacks.

Partial convulsions disappear more quickly after operation than general convulsions. In operating, two kinds of flaps may be employed; without a bone defect, like that of Wagner, and with a bone defect covered by a soft tissue flap. Pieces of bone, foreign bodies, and bone thickening should be removed. Adhesions should be separated between the skin and bone, bone and dura, and dura and brain. The dura may be excised. Cysts and abscesses should be incised and scars excised.

Treatment of Spontaneous Gangrene of the Extremities.—Koga (*Deutsch. Zeitschr. f. Chir.*, 1913, cxxi, 371) says that spontaneous gangrene of the extremities from arteriosclerosis in the young is more common in Japan than in Europe. His chief, Ito, attributed this to the greater use of a vegetable diet in Japan. Until the present time we have been almost powerless to prevent threatening angiosclerotic gangrene, or to restrain it when it develops. The results of arteriovenous anastomosis have not been favorable. In only 8 out of 63 cases collected by Zesas was improvement or a cure obtained. Koga has tested the viscosity of the blood in a series of cases of spontaneous gangrene and found it almost always of much value. Reduction of the increased viscosity by saline infusions influenced the gangrene favorably. The administration of potassium iodide had little or no effect on the gangrene. Koga reports 13 cases, varying in age from twenty-four to forty-eight years. He determined nothing definite as to the etiology, and the urine contained nothing abnormal and particularly no sugar. Syphilis was always excluded, the Wassermann reaction being negative in every case. They were cases of presenile gangrene, in which the viscosity was increased, as a rule, and was reduced to the normal by the saline solution. Simultaneously the symptoms improved, the edematous swelling decreased, as did the cyanosis, and a sharp line of demarcation developed. Ulcers became clean and cicatrized, and the pulsation of the arteries which could not be felt before the introduction of the saline infusion, reappeared. The pain also disappeared so that the patient could sleep, the feeling of cold was not complained of, and the patient could walk a considerable distance. The blood, having its viscosity reduced, gets through the vessels easier and then favors the development of collateral branches. Koga believes that the condition of sclerosis is improved. He reviewed his cases last December, by mail, and found that the improvement or cure had lasted a variable period. In one case amputation had been done for recurrence. In another a black spot had appeared on the same foot. Repeated infusions might have prevented the recurrences. The effect of the infusions on spontaneous gangrene does not appear to be of short duration.

Paralysis of the Phrenic Nerve from the Employment of Kulenkampff's Brachial Plexus Anesthesia.—Seivers (*Zentralbl. f. Chir.*, 1913, xl, 338) reports the following experience following the injection of the brachial plexus above the clavicle for anesthetization of the upper extremity. It was done for the treatment of a wound of the soft tissues on the ulnar side of the right hand, in a healthy young man, and 20 c.c. of a 2 per cent. novocain bicarbonate solution was

injected. The anesthesia was sufficient at the end of a quarter of an hour to permit the operation to be completed without further anesthesia, although the radial side of the hand was not completely anesthetized. At the beginning of the treatment of the wound the patient began to complain of severe pain in the right side of the chest, which increased during the operation. The breathing was inhibited as in a dry pleurisy, every deep inspiration being prevented. The Röntgen rays showed at first almost complete arrest of movement in the right half of the diaphragm. Later there was slight movement of this side, but considerably less than on the left side. The condition remained unchanged for several days except that the pain became less, and the disturbances disappeared in about four days. Three explanations are offered for the development of this paralysis of the phrenic nerve: (1) It may have been due to an endoneural injection of the cervical portion of the phrenic; (2) to a subfascial diffusion of the anesthetic fluid to the nerve; (3) or to an extension of the fluid to the apex of the pleura. Seivers favors the third explanation. The apex of the pleura lies just underneath the brachial plexus and a considerable quantity of the anesthetic fluid is injected, so that it could easily extend along the loose connective tissue over the pleura to the phrenic nerve, which passes downward on the anterior and mesial surface of the pleura. This complication is not, however, a contraindication to this method of anesthetizing the upper extremity. It may be serious, however, in affections interfering with the respiratory organs, since the interference with expiration and coughing might lead to retention of the secretions and pneumonia. Kulenkampff called attention to the possibility of a paralysis of the phrenic nerve in the employment of his method of injecting the plexus.

The Extended Abdominal Radical Operation for Cancer of the Uterus.—WEIBEL (*Surg., Gynec., and Obst.*, 1913, xvi, 251) says that in criticizing the value of the operation we should first calculate how many of all cases subjected to the radical abdominal operation are free from recurrence at the end of five years (after results), and then calculate how many of all cases examined in the clinic (including the inoperable ones) are well and free of cancer at the end of five years (absolute percentage of cures). The after results were calculated as follows: 380 cases were operated, 8 of these died from intercurrent diseases, and 160 were well and free of recurrence; therefore, 43 per cent. of all operated cases could be permanently cured. If the primary deaths were left out of the calculation (because they cannot come into consideration with respect to the after results), we have 53 per cent. permanent cures of all cases surviving the operation. Calculating the absolute efficacy he gets the following results: 863 cases with cancer of the cervix were examined in this five-year period, 36 of them refused the proposed operation, and 8 died from intercurrent diseases; from the rest (819) 160 cases were well and free of cancer. It is 19.5 per cent. absolute efficacy, 1 per cent. more than he had in his first 250 cases. With these results we can say that the radical abdominal operation for cancer of the uterus cured permanently a fifth of all these cases examined in the clinic; it cured permanently 43 per cent. of all operated, and 53 per cent. of all cases surviving the operation. In

the last 175 operations he has been able to reduce the primary mortality to 9 per cent. The raising of the operability, the permanent falling of the mortality, and the increasing in the operative technique allow him to hope that he will be able to report much better results in the next years, when his present cases have passed the five-year limit.

THERAPEUTICS

UNDER THE CHARGE OF

SAMUEL W. LAMBERT, M.D.,

PROFESSOR OF APPLIED THERAPEUTICS IN THE COLLEGE OF PHYSICIANS AND SURGEONS, COLUMBIA UNIVERSITY, NEW YORK.

Syphilitic Disease of the Aorta.—DENEKE (*Deutsch. med. Woch.*, 1913, xxxix, 441) believes mercury is a more reliable remedy for the treatment of syphilitic aortitis than salvarsan or neosalvarsan. He advises giving a combination of mercury and iodide when the Wassermann reaction is positive and iodide alone when the reaction is negative. He says that salvarsan should not be given to patients with serious aortitis and great caution is necessary when giving it to patients with the milder forms of aortic disease. He gives salvarsan only in small doses from 0.2 to 0.4 gram, and always with mercury or the iodides. The guide as to the length of treatment is the Wassermann reaction, and this should become permanently negative before treatment is discontinued. This result may never be obtained, but the treatment should be persistent. Temporary improvement often follows antisyphilitic treatment in cases of aortic disease, but permanent results depend upon the thoroughness of the treatment.

Action of Benzol on Leukemia.—KLEIN (*Wien. klin. Woch.*, 1913, xxiv, 357) has treated 22 cases of leukemia with benzol in the past six months. The details of 12 cases are given in the article. He gave the benzol mixed with olive oil either in milk or in a capsule. His average dose was less than 4 grams a day, and he thinks that the results were equally as good as with higher dosage. Doses as high as 5 grams a day were given in a few cases, and it seemed that these higher doses had an injurious action on the red-blood corpuscles. He also injected the benzol subcutaneously in dosage of 1.5 grams once a day, mixed with an equal amount of olive oil. None of his patient showed any signs of irritation on the part of the kidneys. His results were not so uniformly favorable as those reported by Kiralyfi, Koranyi, and others, but he thinks that benzol is a decided aid in the treatment of leukemia. The best results were obtained when the benzol treatment had been preceded by a course of *x*-ray treatment. He advocates the combination of these treatments in all cases of leukemia, especially for those with a very high leukocyte count. No difference in the effect was noted in the various forms of leukemia.

Treatment of Leukemia with Benzol.—STERN (*Wien. klin. Woch.*, 1913, xxvi, 365) reports a case of leukemia of recent origin treated with benzol. The dose was begun at 3 grams a day in capsules with an equal amount of olive oil and was increased to 6 grams a day. At the end of two months the leukocyte count had diminished from 264,000 to 13,300, and the red-blood cells had increased from 3,500,000 to 5,500,000. The differential count changed as follows: Myelocytes diminished from 44 per cent. to 3 per cent.; polynuclear leukocytes increased from 48 per cent. to 74 per cent. The spleen decreased in size in proportion to the improvement in the blood picture, until at the end of the treatment it was normal in size. The patient's general condition improved, the weight increased, and the result seems to be an apparent cure after two months of benzol treatment. Stern says that of course it is too soon yet to say this is a permanent cure.

Experiences with Neosalvarsan.—LIER (*Wien. klin. Woch.*,1913, xxvi, 410) draws the following conclusions from his experience with neosalvarsan. Neosalvarsan is especially indicated in those cases in which mercurial treatment leads to severe stomatitis or nephritis. In such cases neosalvarsan can be injected repeatedly without periods of interruption of the treatment as is necessary with mercurial treatment. Neosalvarsan may be given intramuscularly because of its easy solubility in water and neutral reaction. The intramuscular injections are very little or not at all painful and they do not leave areas of infiltration at the site of injection. Lier also believes that neosalvarsan can be used with safety in ambulatory cases. He believes that neosalvarsan gives the best results in primary to tertiary syphilis, but that it is also of value in the infectious wet form of the secondary stage. In the dry form of the secondary stage the action of neosalvarsan is weaker than that of mercury, and in these cases it should be used always in combination with mercury. When neosalvarsan is given in combination with mercury the individual injection should be given at longer intervals. Lier believes that neosalvarsan is often able to effect a true abortive cure in primary syphilis if given in large doses at frequent intervals. He also advises a combined neosalvarsan and mercurial treatment in early metasyphilitic diseases of the nervous system.

The End-results of the Abortive Treatment of Syphilis with Salvarsan.—MÜLLER (*Munch. med. Woch.*, 1913, lx, 408) writes concerning 39 cases of primary syphilis treated during 1910 and 1911 by what he terms the abortive treatment of syphilis. This treatment comprises excision or cauterization of the primary lesion, three intravenous injections of salvarsan of 0.4 gram each, and five injections of 40 per cent. calomel in oil (0.5 to 0.07 gram) and 40 per cent. mercurial 0.1 to 0.14 gram. Nine of these cases disappeared from observation, but in the 30 cases observed up to the present time none has shown any clinical manifestation of the disease, and the Wassermann reaction remains persistently negative.

Cholesterin in Paroxysmal Hemoglobinuria.—PRINGSHEIM (*Med. Klin.*, 1913, ix, 254) tried cholesterin in the treatment of paroxysmal

gemoglobinuria on the ground that cholesterin has been found to have an inhibitory action on hemolysis in test-tube experiments. He found that it did abort the attacks and that when it was discontinued the attacks recurred as before. He gave the remedy by intramuscular injections of 5 c.c. at a dose of a 10 per cent. emulsion of cholesterin. No appreciable changes were observed in the blood during the cholesterin treatment. The remedy did not have any permanent effect, however, for a severe and typical attack occurred a week after discontinuing the cholesterin.

The Treatment of Amebic Dysentery with Subcutaneous Injections of Emetin Hydrochloride.—LYONS (*Jour. Amer. Med. Assoc.*, 1913, lx, 1216) reports 6 cases of amebic dysentery treated by hypodermic injections of emetin hydrochloride, 5 of which recovered promptly. Lyons says that the 1 fatal case should be omitted in judging the treatment, as the patient was beyond hope of cure by any method of treatment. The largest dose used was three-fourths of a grain. The average length of treatment until the stools became normal was nine days for the 5 cases. Lyons says that larger doses may be found more effective. There were no ill effects noted from the use of the remedy. He believes the treatment of amebic disease with emetin rests on an experimental basis. It has been shown that ipecacuanha without emetin has but little effect on ameba in vitro (as well as clinically), while emetin has a most powerful amebicidal action. Granting this, then we may assume that emetin is the active principle of ipecac so far as amebas are concerned, and theoretically should be used in preference to the whole drug in the same manner as we employ quinine for the treatment of malaria in preference to cinchona. According to Rogers a third of a grain of emetin is equivalent to 30 grains of ipecac. The soluble salts of emetin are put up by several pharmaceutical houses. The hydrochloride, to which Lyons experience is limited, is practically non-irritating when used subcutaneously, and in moderate doses causes no nausea, vomiting, or depression. Rogers' largest dose was 3 grains in one day. Allen injected 4 grains at one dose and produced nausea for several hours—the patient vomiting once. Rogers has given the hydrochloride intravenously (1 grain in 5 c.c. of normal saline solution) without any depressing effect on the pulse. To summarize, the advantages of the emetin treatment are briefly: (1) Simplicity and ease of administration of the drug; (2) no vomiting or depression; (3) accurate dosage; (4) rapid absorption and effect; (5) reliability of product against the marked differences in the strength of powdered ipecac from different manufacturers. Lyons says that while no definite conclusions can be drawn from the observations of so small a number of cases, he believes that the results are highly suggestive that in the subcutaneous injections of soluble emetin salts an ideal method has been found of treating amebic disease. Time will soon show whether or not, as Rogers believes, another specific has been found.

Amebic Abscess of Liver Treated Successfully by Emetin.—CHAUFFARD (*Bull. d. l'Acad. d. Médicine*, 1913, lxxvii, 122) reports a case of a large amebic abscess of the liver that after about a year per-

forated into a bronchus. When the emetin treatment was begun the patient had been for five months raising a considerable amount of reddish pus averaging each day from 200 to 250 c.c., and during this time had become emaciated and septic. At the same time there was some ulceration in the rectum. The *x*-ray examination showed an opacity at the base of the right lung merging with the shadow of the liver. The patient was given six injections of emetin hydrochloride, each 0.04 gram, during a period of five days. The injections were practically painless and produced no local induration. The expectoration was reduced to but a slight amount on the fifth day of the treatment, and after that it stopped entirely. The temperature fell to normal and the leukocyte count dropped from 49,000 to 19,800, and the polynuclear from 77 per cent. to 63 per cent. The ulceration in the rectum healed and subsequent *x*-ray examinations showed that the base of the right lung had cleared up. The patient improved markedly in general health and increased in weight 13 pounds. Chauffard believes that his experience with the clinical results reported by others demonstrates that emetin is a specific for amebic disease like quinine for malaria.

PEDIATRICS

UNDER THE CHARGE OF

LOUIS STARR, M.D., AND THOMPSON S. WESTCOTT, M.D.,

OF PHILADELPHIA.

Chronic Infective Endocarditis.—EDMUND CAUTLEY (*Archiv. of Pediatrics*, 1913, xxx, 328) offers the histories of a number of cases of this kind, with comments on each. This disease is infrequent in older children and young adolescents, is acute in type, and recovery is exceptional. The following cases are in accordance with the established opinion that infective endocarditis is secondary to pre-existent valvular disease and may affect any valve: An instance of the pyemic type affecting the tricuspid valve occurred in a young adult. After three months in the hospital the patient was discharged cured, except for a permanent tricuspid regurgitant murmur. A case, secondary to mitral valvular disease, in a girl, aged sixteen years. Death occurred from rupture of an aneurysm of a cerebral artery caused by an embolus. A case secondary to rheumatic fever and congenital heart disease in a girl, aged fifteen years. Death occurred from recurrent hemoptysis due to emboli carried to the lungs. A case affecting the aortic valves, probably secondary to a previous heart condition in a boy, aged eight years. Death occurred from meningitis in eleven days, probably from infection carried from the heart. Death results more commonly from secondary effects such as cardiac failure or infarction rather than from toxemia. This is illustrated by two cases. One case of chronic infective endocarditis of the pulmonary valve in a child, aged eight years, was in the hospital eight months. Death was caused

by thrombosis of the pulmonary artery, caused partially by prolonged toxemia and wasting. Fever and wasting were constant and prolonged. The spleen was much enlarged. A reduction in fever occurred with the onset of edema and ascites. Serum and vaccines were valueless. The other case was in a boy, aged eighteen years, in whom the tricuspid and aortic valves were involved. Death was due to a terminal infection of the lower lobe of the left lung and secondary cardiac failure. The spleen was much enlarged but the degree of fever less than in the first case. A streptococcus was found in the blood but autogenous vaccines were useless and the streptococcus was probably due to the terminal infection. Serum and vaccine treatment are practically hopeless, except they may in some cases enable the body to destroy the infective agent if given very early. Quinine, arsenic, iron, and perchloride of mercury are of value.

Diphtheritic Paralysis.—J. D. ROLLESTON (*Archiv. of Pediatrics*, 1913, xxx, 335) reports his findings in a study of 2300 cases of diphtheria with especial reference to diphtheritic paralysis. He found it exceptional for paralysis to develop after the sixth week. Of the 2300 cases, 20.7 per cent. showed some form of paralysis. In each series of 100 cases the percentage of paralysis was never less than 10 or more than 31. He shows that there exists a close relation between the acute attack and the subsequent paralysis. The more severe the acute attack the more frequent and severe the subsequent paralysis. Where the nostrils as well as the throat are affected the incidence of paralysis is greater than when the fauces alone are affected. Pure nasal or laryngeal cases showed practically no subsequent paralysis. Affection of the tendon jerks and the presence of Babinski's sign is more common in severe than in mild attacks. But 1.3 per cent. of the cases showed a relapse and none of these cases developed paralysis. Second attacks, varying from three months to fourteen years, were found in 2.1 per cent. of the cases. Paralysis was more frequent in children than in adults, the majority occurring between two and six years of age. Early injection of antitoxin undoubtedly jugulates the disease, and minimizes the occurrence of complications. Cardiac and palatal paralyses are the only ones occurring during the first fortnight. After this time no serious palsy occurs, as a rule, until the fifth week. During the fifth and sixth weeks other palsies develop, such as ocular, diaphragmatic, etc. In this series 85 deaths were due to paralysis. The prognosis is better in older patients. There was no fatal case above the age of thirteen years. Cardiac, pharyngeal, and diaphragmatic palsies are the only kind which may cause anxiety. A well-marked serum reaction at the usual period after injection is a favorable omen. It is exceptional for cardiac paralysis to be fatal in these cases. In severe cases the patients should not be allowed to sit up for six weeks.

Summer Heat and Summer Diarrhea.—HECTOR CHARLES CAMERON (*British Jour. Child. Dis.*, 1913, x, 205) believes that this subject has been dealt with too exclusively from the point of view of the epidemiologist. All factors in the etiology of summer diarrhea other than microbic contamination have been too much neglected. Even if we could secure a faultless supply of milk we should still

witness an enormous rise in the frequency of diarrhea as a symptom during hot weather. Heat may have a direct effect on the infant, increasing the amount of summer diarrhea. The increase may be due to indirect effect of the heat by lowering the tolerance to food, by increasing the danger of overfeeding in a thirsty infant, by lowering the immunity of the child to infections, and by aggravating the course of all alimentary and infectious disorders of whatever nature. Heat may exert an indirect effect by favoring the multiplication of micro-organisms without the body. If the cause of summer diarrhea is to be found in the multiplication of bacteria in milk, then some explanation must be found for the frequency with which breast-fed infants are attacked and for the high mortality among babies fed on condensed milk and patent foods, in which the bacterial content is relatively low. A vast amount of bacteriological work has not succeeded in establishing a causal relation between any one organism and so-called epidemic diarrhea. In the rise in incidence of summer diarrhea all the factors mentioned have a part, and of all deaths registered as due to diarrhea in the summer, a small fraction only is due to bacterial infection of the alimentary tract. Greater emphasis should be laid on prophylactic measures such as an adequate quantity of water, reduction in carbohydrate food, cooling baths, light, porous clothing, and proper ventilation.

OBSTETRICS

UNDER THE CHARGE OF

EDWARD P. DAVIS, A.M., M.D.,

PROFESSOR OF OBSTETRICS IN THE JEFFERSON MEDICAL COLLEGE, PHILADELPHIA.

Albuminuria Likely to Recur in Successive Pregnancies.—Under this title SLEMONS (*Amer. Jour. of Obstet.*, May, 1913) discusses the question of prognosis in cases which have suffered from albuminuria and toxemia in the first pregnancy. From clinical records it appears that but one in six patients who have had this complication in their first pregnancy may expect its recurrence in subsequent parturition. Lapage (*Annali di ginecologia d'Obstet.*, 1912, p. 577) and Williams, arrive at practically the same conclusion, as among hospital cases 21 per cent. suffered a recurrence of albuminuria and toxemia after the first pregnancy. In the effort to distinguish between those patients who are likely to have danger and those who are not, the clinical course of the two types of auto-intoxication must be studied. A severe attack of scarlet fever in childhood greatly influences the prognosis. Severe toxemia tends to recur in successive pregnancies with increased virulence, and more early in the course of gestation. Where repeated toxemia terminates fatally the kidneys are usually found to be involved. It is sometimes possible to base a prognosis upon the convalescence

of the mother from the first auto-intoxication. If this is prolonged and imperfect, the outlook for the future is correspondingly bad. Slemons would classify cases of albuminuria with toxic symptoms in the later months of pregnancy into three groups; two are well defined and likely to suffer from recurrence, while the third will almost certainly be exempt. He has not found the nitrogen partition to be useful in reaching a prognosis, but he believes that the estimation of the quantity of albumin is helpful. The greater it is and the longer it persists, the more extensively are the kidneys damaged. In cases where the liver seems especially involved and the kidneys normal, the prognosis as to recurrence is good. Blood pressure is more satisfactory than albumin as an index, and upon this basis cases may be divided into three groups. In nephritic toxemia a pressure of 250 m.m. is frequently observed. In toxemia it is at its height; the pressure was rarely below 180 m.m. In cases studied from this basis, 75 per cent. of patients having eclampsia are not likely to do so again; while in 15 per cent. extensive lesions of the kidneys made the prognosis bad; 10 per cent. of toxemic cases are uncertain as to prognosis. Chemical methods in estimating the efficiency of the kidneys have proved disappointing. Slemons concludes that at present we have no better clinical methods for deciding the nature of a toxemia and of reaching an ultimate prognosis in the study of the albumin and blood pressure during convalescence. In nine out of ten cases a satisfactory conclusion can be reached from this data, and auto-intoxication does not return in approximately 80 per cent. of cases.

Abderhalden's Serum Test for Pregnancy.—Behne (*Zentralbl. f. Gynäk.*, 1913, No. 17) has tried Abderhalden's method of dialysis in the diagnosis of pregnancy in the clinic at Kiel. His observations in 44 cases show that this test gives regularly a positive result in normal pregnancy. In advanced gestation, in some cases the results are negative. Patients who are not pregnant but who suffer from an inflammatory condition of the genital organs, with the production of pus or with suppuration in some other portion of the body, as in the breast, often give a positive reaction. In the differential diagnosis between ectopic gestation and inflammatory conditions of the adnexa, the test by dialysis does not give a definite differential diagnosis. Positive reactions are obtained with serum in pulmonary tuberculosis and diseases of the liver.

Puerperal Eclampsia.—Gibbons (*British Med. Jour.*, April 26, 1913) reviews to a considerable extent the literature of the subject and states that we know of nothing which can definitely be described as the cause of the disease, although all evidence goes to show that it is a poison circulating in the blood. Rapid emptying of the uterus by the most safe method available, after the first few convulsions, seems to give the best prognosis. The greater the delay the greater will be the danger to the mother.

Unusual Fertility in Syphilitic Patients, with Anomalous Involvement of the Child.—Watson (*British Med. Jour.*, April 26, 1913) reports the remarkable instance of the family of a wandering gypsy,

the mother of the family having brought one of her children for examination. Multiple dactylitis was present, of syphilitic origin. On examination, the mother was aged twenty-one years, well nourished, and gave a negative history, believing that she had never had venereal disease. On examination, there was a scar on the right labium. She had three times given birth to twins, without miscarriages. All of the children were born prematurely, the first pair at the seventh month, and the others at the eighth month. The first twins were normal in size, the others smaller. The father had undoubtedly had syphilis, and was fairly vigorous, but was mentally deficient and seemed to be in the very early stage of general paralysis of the insane. The first twins seemed in perfect health and showed no evidence of syphilis. The second pair had thickening in the bones and dactylitis, and the parents stated that the children had eruptions on the buttocks when they were six weeks old. The third twins had a condylomas, sore eyes, and peeling of the skin of the hands and feet. There were also marked snuffles and a coppery eruption on the nates. The serum of the parents and children in each case gave a very positive result. This family of gypsies were wandering about the country subsisting merely by selling tin cooking utensils which they made. They are a source of danger from infection and such individuals should be segregated to prevent the danger of spreading syphilis in the community.

The Condition of the Blood Serum of Mother and Fetus in the Pyelitis of Pregnancy.—Weivel (*Archiv f. Gynäk.*, 1913, cxix, No. 2) has studied the question of the formation of antibodies in the blood serum of mother and fetus in cases where pregnancy was complicated by pyelitis. He finds that the colon bacillus passing from the intestine into the urinary tract takes up its new functions and activities from the antibodies, and what he styles the third order may be detected in these cases; the reactions are not always typical, and the organisms having immunity show in various cases great differences, which complicate the results of clinical and bacteriological examination. When antibodies were found in the serum of the mother, they were also present in the serum of her child or children. In some cases they were also present in the amniotic liquid in a greatly lessened quantity. In the serum of the fetus these bodies disappeared shortly after birth. It is evidently not yet practical to employ this reaction in making a diagnosis of infection by the colon bacillus.

Subcutaneous Symphysiotomy.—Kehrer (*Archiv f. Gynäk.*, 1913, xcix, No. 2) reports 10 cases of subcutaneous symphysiotomy, which he performs under ether or chloroform anesthesia, or in some cases by spinal anesthesia. Under antiseptic precautions two fingers of the left hand are inserted in the vagina and the urethra carried to one side. The clitoris and meatus are pushed, so far as possible, out of the way, and an incision made directly with the scalpel upon the middle of the symphysis. The under portion of the symphysis is separated through two-thirds of the joint. The edge of the knife is then turned upward and the upper portion of the joint severed, while the separation is made complete in the surrounding tissues by the

use of a smaller scalpel. When the ligamentum arcuatum is severed the pubic bones separate, and the legs of the patient are rotated outward and somewhat inward by assistants, to prevent the laceration of tissue. The wound is then packed with gauze and the extremities closed with metal clips. The bladder is then carefully emptied completely by catheter, and the patient placed in bed, when pituitrin in some form is given by intramuscular injection. The upper portion of the patient's body is slightly raised, the knees are brought together, the thighs bandaged, and the knees slightly separated as birth occurs. After the birth of the placenta a permanent catheter is placed in the bladder for several days. In 118 cases the maternal mortality was 0.8 of 1 per cent., in comparison with 4.1 per cent. in 217 subcutaneous hebostiotomies. There was no fetal mortality. There is almost no hemorrhage in the operation, the wound through the skin is exceedingly small, and the danger of wounding the surrounding tissues is very slight. In 60 per cent. of cases there was some injury to the crura of the clitoris, and hematoma formed; and in 30 per cent. of the cases thrombophlebitis complicated the puerperal period. No case of embolism was observed. The operation finds an especial field in multiparæ, and the best results are obtained when the child is expelled spontaneously. If possible, delivery by operation should be avoided, and some preparation of pituitrin, with possibly Walcher's position, should be utilized for delivery. Uterine inertia is no contraindication to the operation, and it may be performed in pelves whose true conjugate is as low as 6.9 to 6.8 cm. If care is exercised in delivery and in separating the pelvis, the sacro-iliac joints may not be damaged, and a separation of the pubic bones greater than 3 cm. should not occur. The operation is indicated where cases are suspected of infection. The patients walk well after the operation, and infection does not occur more often than in other cases. The joints unite as well as does the pelvis after pubiotomy, and the pelvis remains somewhat enlarged after the operation. No callus forms, as after hebostiotomy, and the permanent scar is very small. The one essential objection to the operation is the tendency for hematomas to form in tissues about the severed ends of the joint.

GYNECOLOGY

UNDER THE CHARGE OF

JOHN G. CLARK, M.D.,

PROFESSOR OF GYNECOLOGY IN THE UNIVERSITY OF PENNSYLVANIA, PHILADELPHIA.

Intestinal Obstruction Due to Retroverted Uterus.—A case of acute intestinal obstruction due to the sudden incarceration of a loop of intestine behind a non-adherent, retroverted uterus is reported by LEFÉVRE (*Jour. de Méd. de Bordeaux*, 1913, xliii, 183). The patient had had for some years a markedly retroverted uterus, which, however,

gave only slight symptoms, until one day, when riding in a carriage, she received a severe jolt, following which she was instantly seized with violent abdominal pain. This continued, and soon vomiting came on, which later became fecal. Nothing was passed by the bowel for forty-eight hours, by which time the patient was in a critical condition, presenting all the classical symptoms of intestinal obstruction. Laparatomy revealed a collapsed cecum and large intestine, but the small intestine was enormously distended up to a point where a loop dipped down into Douglas' pouch behind the uterus; beyond this it also was collapsed. Gentle traction was sufficient to bring the intestinal loop out of the small pelvis, when there was an immediate passage of gas and fecal matter into the collapsed portion of the gut, and the condition was relieved. The patient made an uneventful recovery. Lefèvre believes that at the moment the patient received the jolt in the carriage the uterus was bounced forward for a moment out of its position of extreme retroversion, giving a loop of intestine the chance to slip down behind it. This was then caught by the uterus dropping back against the sacrum, and as distention began to take place behind the point of impingement, intra-abdominal pressure was increased, and the caught loop of intestine still further compressed. The uterus was not pregnant, and was only slightly larger than normal.

Prognostic Value of Leukocyte Count in Pelvic Suppurations.—A statistical study of 100 cases of suppurative conditions in the pelvis from Hunter Robb's service in Cleveland has been made by J. T. SMITH (*Surg., Gynec., and Obst.*, 1913, xvi, 403) to determine if the pre-operative leukocyte count is of any value in forecasting the outcome of the case. Of course, in cases where the leukocytosis, fever, and other symptoms more or less correspond, that is, with high leukocyte count and high fever, or with low count and slight fever, this point is not of especial significance, but it does furnish us, Smith thinks, with valuable data in that group of cases in which the blood count and temperature record are at variance, that is, in patients with a high leukocytosis (over 14,000) and moderate fever (below 101°), or conversely, with a low count (below 14,000) and high fever (above 101°). In these instances Smith thinks the blood count is of more prognostic value than the temperature, since the recorded results in the 100 cases studied show that some trouble arose in a majority of the cases of pus in the pelvis with a leukocytosis of above 14,000, whereas only a small proportion of similar cases with a count below 14,000 developed any postoperative complications; moreover, the mortality in the first group was 8 per cent., as opposed to zero per cent. in the second.

Callous Ulcer of the Bladder.—BUERGER (*Med. Record*, 1913, lxxxiii, 657) says that within the past two years he has seen 2 women suffering with intense vesical tenesmus, dysuria, great frequency and urgency of micturition, and pyuria, in whom the cystoscope revealed the cause of the trouble to be a solitary, callous ulcer of the trigone, a condition as to whose occurrence doubt has been expressed by a number of prominent urologists. In one of these cases fulguration was tried, but entirely without success, and the author believes that where the

process is deep-seated, has lasted for a long time, with the formation of phosphatic incrustations on the surface, neither the application of silver nitrate nor cauterization will have any beneficial effect. The symptoms are so intense as to demand in every case radical measures for their relief, and Buerger has devised a method of treatment which in the two reported cases was followed by excellent results. He introduces through the operating cystoscope a pair of flexible "punch-forceps" with cup-shaped jaws, directing them against the lesion in much the same way as an ureteral catheter. In each case the entire ulcer was excised by means of these forceps at one sitting, cutting well down into the underlying bladder wall; this procedure was followed by immediate amelioration of the symptoms, the condition rapidly progressing to complete cure. Subsequent cystoscopic examination showed that perfect healing of the ulcer had taken place with complete restoration of the vesical mucosa. Pathological examination of the tissue removed showed a superficial deposit of urinary salts, then a layer of necrosis and ulceration, and beneath this newly-formed connective tissue with active evidences of inflammation.

DISEASES OF THE LARYNX AND CONTIGUOUS STRUCTURES

UNDER THE CHARGE OF

J. SOLIS-COHEN, M.D.,

OF PHILADELPHIA.

Three Fatal Cases of Pneumococcal Infection of the Throat.—TWEEDIE (*Jour. Laryn., Rhinol., and Otol.*, April, 1913) places on record the details of a clinical condition which he had not otherwise seen, the mortality of which in his hands had been 100 per cent. Two patients represented the asthenic, and a third the sthenic type of the disease, the latter running a comparatively rapid course from its commencement up to the end. All three were characterized by their absolute lack of response to every kind of general or local treatment employed; and no cause or connection could be ascertained apart from the presence of the pneumococcus in the throat which was present in large numbers in pure culture on the swabs or sputa. There was no apparent ulceration of, nor membranous deposit on the mucous membrane concerned, which was, however, intensely injected and edematous at the commencement of the illness. The main focus of the local lesion in each case appeared to the naked eye as an indolent, necrotic, more or less localized, sloughing area quite distinct from any pathological state usually associated with the term "abscess." Up to the last few days of the disease there was no impairment of the appetite or malaise corresponding with the progressive weakness, raised temperature, or severity of the disease generally.

Abnormal Epistaphylian Tonsil.—DAPITOLO reports (*Annales des Maladies de l'Oreille, du Larynx, du Nez et du Pharynx*, March 3, 1913) a case in a child, aged seven years, upon whom he performed a bilateral palatine tonsillectomy with adenectomy. Three months afterward he removed, with a cold snare, a tumor of the size and form of a large molar tooth inserted by a short pedicle, upon the superior surface of the soft palate. Histological examination showed that it was composed of lymphoid tissue, and Dapitolo believes that it was an adenoid hypertrophy, provoked by irritation from the operation.

A Case of Mixed Tumor of the Soft Palate.—ALAGNA (*Annales des Maladies de l'Oreille, du Lar., du Nez et du Phar.*, March 3, 1913) reports a man, aged fifty-five years, who had a voluminous tumor of the soft palate, with troubles in deglutition and nasal respiration. Histological examination showed that it was composed of a lipo-myxomatous tissue, the adipose cells of which were constituted by a mixture of ethers, glycerin, and cholesterine.

Perforation of the Nasal Septum Due to Topical Action of Cocaine.—CHEVALLIER (*Revue Hebd. de Lar., d'Otol. et de Rhinol.*, April 26, 1913) presents a thesis on the subject of perforation of the septum due to the sniffing of cocaine which he has frequently observed in individuals who might be classed among degenerates, or who have lost their equilibrium. He has also noticed it very frequently among morphomaniacs. This perforation is always localized upon a point of the septum corresponding with the cartilaginous skeleton, the quadrangular cartilage alone being involved in the necrosis. As to its etiology, the author contends that the leukocytes are killed by the cocaine and become harder, and thus they clog and gradually obliterate the lumen of the capillaries, rendering definitive a local anemia which is originally produced temporarily. This anemia long kept up proceeds to a suppression of nutrition and to the mortification of the tissues which ultimately terminates in the necrosis. An eschar is produced, and its elimination is the first stage in the evolution of an ulceration.

Chondrosarcoma of Nasal Passages.—MR. HERBERT TILLEY (*Jour. Laryn., Rhinol., and Otol.*, April, 1913) presented to the Laryngological Section of the Royal Society of Medicine two remarkable photographs of a young lady of sixteen, with recurrence within a few weeks after recovery from a primary operation, and a second recurrence after a second operation for involvement of the nasal septum and right ethmoidal regions.

Bronchial Asthma Cured by Operations in Rhinopharyngeal Respiratory Tract.—GOGOMANN (*Annales des Maladies de l'Oreille, du Lar., du Nez et du Phar.*, January, 1913) reports the case of a man, aged thirty-five years, a subject of asthma. After destruction of a nasal synechia and of a deviation of the septum, the paroxysms of asthma were arrested; and they disappeared completely after ablation of tonsils containing caseous accumluations in the crypts.

Topical Anesthesia in Sinus Operations.—In a discussion on the technique of operations for perinasal sinusitis, SIEBENMANN (*Annales des Maladies de l'Oreille, du Lar., du Nez et du Pharynx*, January, 1913) affirmed that local anesthesia predisposes to local suppuration, and impedes cure by first intention. Ritter reported that he had had a case of death seven hours after an operation as a result of anesthesia by morphine-scopalamin injection.

HYGIENE AND PUBLIC HEALTH

UNDER THE CHARGE OF

MILTON J. ROSENAU, M.D.,

PROFESSOR OF PREVENTIVE MEDICINE AND HYGIENE, HARVARD MEDICAL SCHOOL, BOSTON, MASS.,

AND

MARK W. RICHARDSON, M.D.,

SECRETARY OF THE MASSACHUSETTS STATE BOARD OF HEALTH.

Chlorinated Lime in Sanitation.—Chlorinated lime or bleaching powder, popularly miscalled "chloride of lime," is a very remarkable substance which is being used very widely, especially for the disinfection of drinking-water supplies. The subject is summed up in a book entitled *Chloride of Lime in Sanitation*, by ALBERT H. HOOKER (John Wiley & Sons, 1913). Ballard, in 1835, found chlorinated lime to be composed of: Calcium hypochlorate, $Ca(OCl)_2$; calcium chloride, $CaCl_2$; calcium hydroxide, $Ca(OH)_2$. Subsequent investigations by Olding and other chemists have shown that calcium chloride and calcium hypochlorite do not exist as such in dry bleaching powder, but are found on dissolving it in water. Calcium oxychloride, $CaOCl_2$, is generally accepted to be the essential constituent of dry chlorinated lime, and calcium hypochlorite to be the active principle of the solution. It is also now well understood that chlorinated lime in its industrial application of bleaching, deodorizing, or disinfecting does not act by its chlorine, but by its oxygen. The chemical action is not "chlorination" but "oxidation." Hypochlorous acid (HOCl) is probably the most powerful oxidizing agent known to chemists, and is readily broken up into hydrochloric acid, and liberates nascent oxygen with extreme readiness. Hooker gives convenient methods for making standard solutions of chlorinated lime, with useful tables, comparing parts per million with grains per gallon, expressed in terms of bleach and in terms of available chlorine. As a practical process the use of chlorinated lime for the disinfection of water dates from 1908, when Mr. G. A. Johnson was called to remedy some serious trouble in the water purification at the Chicago stock yards. Filtration of the water of Bubbly Creek was not satisfactory, and Mr. Johnson substituted chlorinated lime for the copper sulphate which had been used. Chlorinated lime is now used by many municipalities to render their public water supply safe. In some places it is used in

conjunction with alum coagulation and sedimentation or slow sand filtration; in fact, it may be used either alone or as an accessory to other methods. Following is a partial list of cities now using hypochlorite of lime in their drinking water supplies: New York, 16 pounds per million gallons; Omaha, 7½ pounds per million gallons after coagulation and sedimentation; Cincinnati, 5 to 12½ pounds—typhoid rate reduced to 5.7; St. Louis; Minneapolis, after mechanical filtration; Toronto, 6 pounds; Montreal, 5 to 7½ pounds; Cleveland, 16 pounds; Erie, 7 to 10 pounds; Chicago; Milwaukee, 6 pounds; Pittsburgh, 3 pounds, after slow sand filtration; Jersey City, 5 to 8 pounds; Council Bluffs, 15 pounds, following alum precipitation; Brainerd, Minn.; Ridgeway, N. J.; Corning, N. Y.; Nashville, Tenn., 14 pounds; Grand Rapids, Mich.; Little Falls, N. J.; Harrisburg, Pa.; Baltimore, Md.; Niagara Falls; Toronto; Ottumwa, Iowa, and others. It is important to remember that in waters treated with chlorinated lime, free chlorine never is present. While the chlorinated lime kills bacteria in the amounts used, it does not purify organic matter, cure discoloration, turbidity, or neutralize tastes and odors in the water. The remarkable germicidal power of chlorinated lime is better understood when it is known that three grains of a practically harmless substance will kill myriads of bacteria contained in a barrel of water. Ordinarily the amounts used are from one to two or five parts per million parts of water. Much more is required in sewage. A bacterial reduction of 99 per cent. may be obtained in a water containing little organic matter, with one part per million, whereas it requires 1 part to 25,000 parts of sewage to affect a similar bacterial reduction. In practice, the average amount of bleach used in water purification is from 5 to 12 pounds per million gallons of water. The bleach cannot be detected by the sense of taste, provided the amount does not exceed 25 pounds. While the chlorinated lime treatment of water supplies is essentially simple, yet it requires able professional supervision else disappointing results will come from haphazard work. The great essential is a uniform dosing of a standard solution. Bleaching powder is also used in the disinfection of the water of swimming pools, for street sprinkling, and flushing, for the disinfection of feces and sputum; and to a certain extent, for the disinfection of glassware, fabrics, brushes, and combs. It is one of the best substances we have for the general disinfection of rough places, such as slaughter-houses, bake-houses, dairies, outhouses, cellars, and the like. In surgery chlorinated soda is used, the action of which is entirely analogous to chlorinated lime.

The Stable Fly as a Carrier of Disease.—Schuberg and Kuhn (*Arbeiten a. d. Kaiserl. Ges.-Amt.*, 1912, xl, No. 2) have studied the question of the transmission of various infections, particularly relapsing fever, anthrax, Southwest African horse sickness (Pferdesterbe), and epithelioma of fowls (Hühnerpocken) through the agency of the stable fly (*Stomoxys calcitrans*). The first report of these studies was published a year ago (*Arbeiten a. d. Kaiserl. Ges.-Amt.*, 1911, xxxi, No. 2) with special reference to trypanosomes and spirochetes. The successful results obtained by Schuberg and Kuhn now have an added significance in view of the recent findings which incriminate the stable

fly as a transmitter of the virus of poliomyelitis (Rosenau, Brues, Richardson, *et. al.*, confirmed by Anderson and Frost). Schuberg and Kuhn successfully transferred the Spirochæta obermeieri from infected rats to healthy rats through the bites of Stomoxys calcitrans. Practically every one of the flies were able to transfer the infection provided only five minutes intervene between the bites of the infected and the healthy animal. If ten minutes intervene some of the flies are no longer infective, and if fifteen minutes intervene the number of infective flies is markedly diminished. It developed, as a result of ten experiments, that infection did not occur after an interval of thirty minutes. Nattan-Larrier, in 1911, showed that the spirochetæ of relapsing fever may live in the intestinal tract of the ordinary house fly that has fed upon infectious material, and pointed out the possibility of thus transferring the infection. Prior to Nattan-Larrier's work Sergent and Foley showed that house flies (Musca domestica) which fed upon the blood coming from the nose of the patient, contained numerous spirochetes which remained intact to microscopic examination at least twenty-four hours. Experiments designed to transfer the infection from man to apes by means of the common house fly were negative. (Other insects, such as body lice, have been incriminated as carriers of the infection of relapsing fever.) Copeman, Howlett, and Merriman, in 1911, showed by coloring flies that they may fly 1700 yards from the place where they are born. It was also shown that a fly may travel about one-fourth of a mile in half an hour. It is therefore evident that a fly such as Stomoxys calcitrans, which is strong upon the wing, may convey the infection of relapsing fever for a distance of about 750 feet during the fifteen minutes it remains infective. The experimental investigations upon Southwest African horse sickness (Pferdesterbe) were likewise successful in that this infection was transmitted through the bites of the stable fly. Schuberg and Kuhn are careful to note that stall infection is possible, and are in some doubt as to how much of a role the Stomoxys plays in nature. These same authors made three experiments by interrupting the bites of Stomoxys in epithelioma of fowls (Hühnerpocken). All three experiments were positive. Schuberg and Kuhn express the opinion that, in view of the great resistance of this virus, especially against drying, and further, in view of the small amount necessary to reproduce the disease, it is not unlikely that Stomoxys may remain infective a long period of time, and it therefore seems that this may be of practical importance in the transmission of the disease in nature. The Stomoxys breeds in manure, and it would, therefore, have ready access to chickens, which they are known to bite under natural conditions. Finally, Schuberg and Kuhn succeeded in transferring anthrax to mice and guinea-pigs through the bites of Stomoxys calcitrans. These experiments consisted in permitting the flies to feed upon the livers and spleens of infected animals, and the longest interval between such feeding and the bites of the susceptible animal was two hours and ten minutes. In one case successful infection was obtained by the bite of one single fly at an interval of ten minutes. Six experiments were then undertaken to transfer the infection from animal to animal with one positive result. The donor was a mouse having large numbers of anthrax bacilli in its blood at the time of the bite; the recipient was a guinea-pig which was bitten ten minutes

later. It is known that anthrax bacilli appear in the circulating blood suddenly, and in nature flies may obtain the infection in this way or from shed blood. The latter perhaps would be more dangerous on account of spore formation. It is evident that Stomoxys cannot play the major role in the transfer of anthrax in nature, but doubtless is responsible for occasional cases, especially anthrax of the skin. Man, as well as animals, may thus receive the infection.

The Role of the Stable Fly in the Transmission of Surra.—MITZMAIN, M. B. (*Philippine Journal of Science*, December, 1912, vii, 6 p. 475) reports the results of a long series of carefully conducted experiments to determine the role of Stomoxys calcitrans in the transmission of Trypanosoma evansi. Negative results were obtained in attempts at direct mechanical transmission of surra with flies, which were induced to bite healthy animals at intervals ranging from five minutes to three days, after being permitted to complete the feeding upon infected animals. Thousands of Stomoxys calcitrans were employed in twenty-nine experiments, involving the use of three horses six monkeys, and twenty-two guinea-pigs. Attempts also proved negative to transmit surra by the interrupted method of feeding. In these experiments the intervals between feeding on infected and healthy animals averaged twenty-five to forty seconds. The only positive result obtained was produced from a succession of 206 interrupted bites, in which the flies were transferred immediately from the infected to the "clean" animal. The flies were applied thirty-two hours during a period of six days. Mitzmain concludes that these experiments indicate that Trypanosoma evansi does not develop in the body of Stomoxys calcitrans. He was unable to find the organisms of surra in the flies beyond eighteen hours after feeding on an infected animal, and the limit for infection by inoculation was six hours. Pathogenic trypanosomes were found in the proboscis of the fly thirty seconds after feeding on infected blood. However, after one minute and thirty seconds the organisms were not present in the mouth parts in a form capable of infecting by inoculation into guinea-pigs. Mitzmain also found as a result of these experiments, that the wounds made by Stomoxys calcitrans are not suitable channels for infection. No evidence was obtained to indicate that Trypanosoma evansi is hereditarily transmitted to the offspring of Stomoxys calcitrans. The larva of this fly fed on surra blood does not continue to harbor the trypanosoma, and the fly is "clean" upon reaching maturity. Mitzmain believes the the individual glass-tube method is the most suitable for applying flies in feeding on experimental animals, and for keeping flies for long periods under laboratory condtions.

Notice to Contributors.—All communications intended for insertion in the Original Department of this JOURNAL are received only *with the distinct understanding that they are contributed exclusively to this* JOURNAL.

Contributions from abroad written in a foreign language, if on examination they are found desirable for this JOURNAL, will be translated at its expense.

A limited number of reprints in pamphlet form, if desired, will be furnished to authors, *provided the request for them be written on the manuscript.*

All communications should be addressed to—

DR. GEORGE MORRIS PIERSOL, 1927 Chestnut St., Phila., Pa., U. S. A.

VOL.

A
T

EXPER
OF
THE
FRC
AN E
SAL
PINCH
By
THE
PIT
TUBE
DO
E.
THE
TH
CONG
A CO
FIL
THE
M.
THE
S.
AN
PR

Phi

COPYRI